"A study on the various etiologies and management strategies in Mechanical obstruction of the Intestine"

Dissertation submitted in
Partial fulfilment of the regulations required for the award of
M.S. DEGREE
In
General Surgery Branch - I

THE TAMILNADU
DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI
APRIL, 2013.

CERTIFICATE

This is to certify that this dissertation titled ***"A study on the various etiologies and management strategies in Mechanical obstruction of the Intestine"*** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of M.S Degree Branch - I (General Surgery) is a bonafide work done by Dr. Anantha Krishna M A, post graduate student in General Surgery under my direct supervision and guidance during the period of September 2011 to November 2012.

Prof.S.Natarajan, M.S.
Professor of Surgery
Dept. of General Surgery
Coimbatore Medical College Hospital

Prof. P.V. Vasantha Kumar, M.S.
Professor and Head of the Department
Dept. of general Surgery
Coimbatore Medical College Hospital

Dr. Vimala, M.D.
Dean,
Coimbatore Medical College Hospital

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : DR. ANANTHA KRISHNA . M.A.

Course : M.S. GENERAL SURGERY

Period of Study : SEPTEMBER 2011 - NOVEMBER 2012

College : COIMBATORE MEDICAL COLLEGE

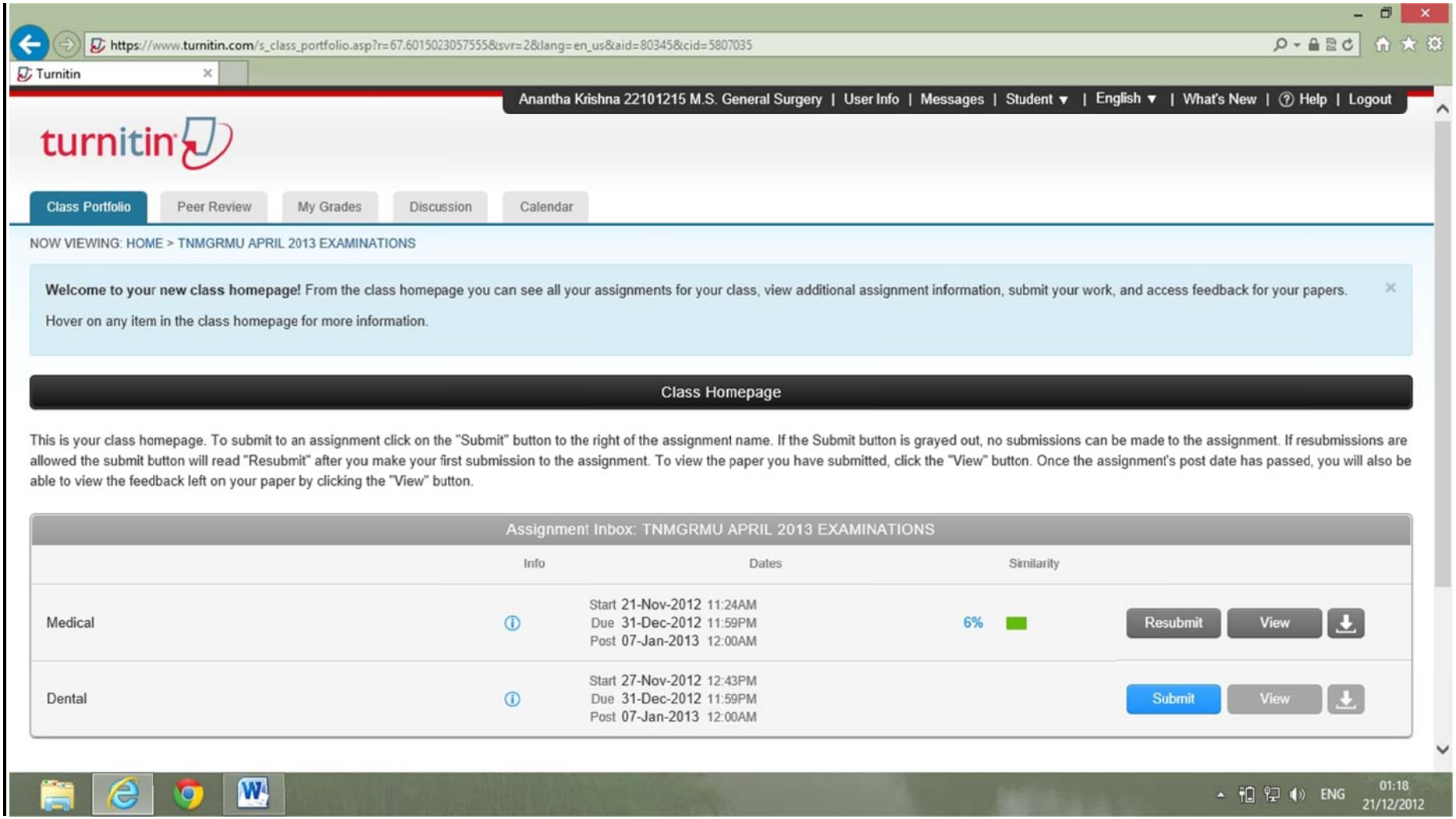
Dissertation Topic : A STUDY ON THE VARIOUS ETIOLOGIES AND MANAGEMENT STRATEGIES IN MECHANICAL OBSTRUCTION OF THE INTESTINES

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 23.9.11

Secretary
Ethics Committee


https://www.turnitin.com/s_class_portfolio.asp?r=67.6015023057555&svr=2&lang=en_us&aid=80345&cid=5807035
Turnitin
Anantha Krishna 22101215 M.S. General Surgery | User Info | Messages | Student ▼ | English ▼ | What's New | Help | Logout
turnitin
Class Portfolio
Peer Review
My Grades
Discussion
Calendar
NOW VIEWING: HOME > TNMGRMU APRIL 2013 EXAMINATIONS
Welcome to your new class homepage! From the class homepage you can see all your assignments for your class, view additional assignment information, submit your work, and access feedback for your papers.
Hover on any item in the class homepage for more information.
Class Homepage
This is your class homepage. To submit to an assignment click on the "Submit" button to the right of the assignment name. If the Submit button is grayed out, no submissions can be made to the assignment. If resubmissions are allowed the submit button will read "Resubmit" after you make your first submission to the assignment. To view the paper you have submitted, click the "View" button. Once the assignment's post date has passed, you will also be able to view the feedback left on your paper by clicking the "View" button.
Assignment Inbox: TNMGRMU APRIL 2013 EXAMINATIONS
Info
Dates
Similarity
Medical
Start 21-Nov-2012 11:24AM
Due 31-Dec-2012 11:59PM
Post 07-Jan-2013 12:00AM
6%
Resubmit
View
Dental
Start 27-Nov-2012 12:43PM
Due 31-Dec-2012 11:59PM
Post 07-Jan-2013 12:00AM
Submit
View
ENG
01:18
21/12/2012

turnitin

Your digital receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

Paper ID	294086176
Paper title	A study on the various etiologies and management strategies in Mechanical obstruction of the Intestine
Assignment title	Medical
Author	Anantha Krishna 22101215 M.S. General Surgery
E-mail	drananthkrishna@gmail.com
Submission time	20-Dec-2012 08:32PM
Total words	16137

First 100 words of your submission

"A study on the various etiologies and management strategies in Mechanical obstruction of the Intestine" Dissertation submitted in Partial fulfilment of the regulations required for the award of M.S. DEGREE In General Surgery Branch - I THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI APRIL, 2013. CERTIFICATE This is to certify that this dissertation titled "A study on the various etiologies and management strategies in Mechanical obstruction of the Intestine" submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of M.S Degree Branch - I (General Surgery) is a bonafide work done by Dr. Anantha Krishna M A, post graduate...

ACKNOWLEDGEMENT

I express my gratitude to **Dr. R.Vimala, M.D.,** Dean, Coimbatore Medical College Hospital for permitting me to use the clinical material for the study.

It gives me immense pleasure to express my deep sense of gratitude to my Unit Chief **Prof. Dr. S. Natarajan, M.S.** General Surgery, Department of General Surgery, Coimbatore Medical College Hospital, for his excellent guidance and valuable suggestions during the course of study and in preparation of this dissertation.

I am grateful to **Prof. Dr. P.V.Vasantha Kumar, M.S.,** Professor and Head of the Department of General Surgery, Coimbatore Medical College Hospital, for his guidance throughout this study.

I also thank the former head of the department of surgery, **Prof. Dr. Mohan, M.S. and Prof. Dr. Nanjundappan, M.S.**

I also express my heartfelt thanks to the unit chiefs, **Dr.Elango, M.S., Dr.Swaminathan, M.S., Dr.Ranganathan, M.S. , Dr.Ravindran M.S., and Dr.Saradha, M.S.** for their suggestions at the apt time that has helped me in the completion of this work.

I am deeply indebted to my Assistant Professors, **Dr.S. Durairaj, M.S., Dr. Meena, M.S., Dr. A. Nirmala, M.S.,** for their help and guidance throughout this study.

I express my thanks to my friends and all others who have helped me in the preparation of this dissertation.

Last but not the least; I heartily thank all the patients for their kind support without whom this study could never be done.

DECLARATION

I hereby declare that the dissertation entitled ***"A study on the various etiologies and management strategies in Mechanical obstruction of the Intestine"*** was done by me at Coimbatore Medical College Hospital Coimbatore – 641018 during the period of my post graduate study for M.S. Degree Branch-1 (General Surgery) from 2011 to 2012.

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the University regulations for award of M.S., Degree in General Surgery.

Dr. Anantha Krishna M A
Post Graduate Student
M.S. General Surgery
Coimbatore Medical College Hospital

CONTENTS

LIST OF TABLES

LIST OF COLOUR PICTURES

ABSTRACT

Background:

The etiological factors of mechanical intestinal obstruction which is one of the common surgical emergencies, varies widely over geographical regions and has been changing over time. In this study, we aim to analyze the epidemiology and outcome of mechanical intestinal obstruction in adults

Materials & Methodology:

A prospective observational study was done at Coimbatore Medical College Hospital, Coimbatore from 2011 to 2012. Inclusion criteria were adults with mechanical obstruction of intestines. Clinical features and findings at laparotomy were tabulated and analyzed.

Results :

A total of 154 patients with mechanical intestinal obstruction who were surgically treated were included in the study. Of these, 68.83% presented with acute obstruction and 31.17% with subacute obstruction. Males (69.84%) outnumbered females (30.16%) and the mean age at presentation was 48.88 years. External hernias getting obstructed (49.34%) was the most common cause followed by adhesive intestinal obstruction (33.76%). Strangulating obstruction with compromise of gut

vascularity was present in 23.37% while the remaining 76.63% was non-strangulating obstruction. A total of 29.87% of the patients developed post-operative complications comprising with wound infections (19.48%). Mortality rate in the study population was 3.8%.

Conclusion:

External herniae were found to be the most common cause of mechanical intestinal obstruction in our study group. This study demonstrates that the pattern of intestinal obstruction in our study population was different than most western studies and emphasizes the need for early diagnosis and prompt treatment.

Key words:

Mechanical Intestinal Obstruction; Dynamic Intestinal Obstruction; Strangulating obstruction; Obstructed/ Strangulated Hernias; Adhesive Intestinal Obstruction

INTRODUCTION

Definition: Bowel is said to have been obstructed when the normal passage of Intestinal contents does not occur [7]

Intestinal Obstruction is one of the most common causes of the 'Surgical Abdomen'. It continues to be a major cause of morbidity and mortality worldwide. The disease is perhaps as ancient as mankind. One of the earliest known records of treatment for this dreaded condition is when Praxagorus Circa in 350 B.C. created an entero-cutaneous fistula to relieve the obstruction of a segment of bowel. With newer methods in diagnosis such as the sophisticated radiological investigations and progress in treatment strategies such as newer and more powerful antibiotics, fluid therapy, parenteral nutrition, most recently transplantation, the incidence of complications has greatly come down. Despite all the recent advances in the diagnostic and management techniques, Intestinal Obstruction shall continue to be a great challenge to the medical fraternity. Prompt recognition and early aggressive treatment helps to reduce the morbidity and mortality associated with this disease.

AIMS & OBJECTIVES

1. To determine the relative frequency of various causes of Intestinal Obstruction
2. To study the various patterns of presentation of Intestinal Obstruction and the diagnostic modalities in Intestinal Obstruction
3. To study the Morbidity and mortality associated with Intestinal Obstruction
4. To study the management of Intestinal Obstruction

REVIEW OF LITERATURE

ANATOMY [1, 2, 5, 6]

Small intestine extends from the pylorus to the caecum measuring about 290 cm. it is divided into three parts, namely –

1. Duodenum from pylorus to ligament of Trietz for a length of 20 cm.
2. Jejunum starts from duodeno-jejunal flexure for about 110 cm and
3. Ileum for about 160 cm upto caecum.

Colon and rectum together contribute a length of 150 cm. this is also further divided into various segments as noted below.

1. Caecum measures about 10 cm length and 7.5 cm diameter. It has a maximum distensibility of 12 cm beyond which ischemic necrosis and perforation may result.
2. Ascending colon runs upwards for about 15 cm and turns at the hepatic flexure into the transverse colon for 45 cm.
3. Transverse colon is completely enclosed in the visceral peritoneum and may be floppy in many patients due to its mobility. It extends for a length of about 45cm.
4. Descending colon runs downwards for 25 cm from the splenic flexure and continues into the thicker and mobile sigmoid colon for a variable length of 15 to 50 cm.

5. The sigmoid colon rests on a long floppy mesentery which makes it prone for volvulus. This continues into the rectum for 12-18 cm which finally ends into the anal canal.

Developmentally, the entire gastrointestinal tract is divided into three segments namely –

1. Foregut from which proximal areas of GIT are derived from the esophagus upto the second part of duodenum. This is supplied by the coeliac axis
2. Midgut gives rise to the distal duodenum, jejunum, ileum, caecum, ascending colon and the proximal transverse colon. This is supplied by the superior mesenteric artery.
3. Hindgut develops into the distal part of the transverse colon, descending colon, sigmoid, and the rectum. This is supplied by the inferior mesenteric artery.

Extending for such a length, the integrity and continuity of the lumen assumes prime importance for the normal functioning. Any alteration in the continuity leads to intestinal obstruction.

Blood Supply

Almost all of the small intestine derives its supply from the superior mesenteric artery except the duodenum which is supplied by the coeliac axis. All vessels supplying the small bowel course through the mesentery and form a rich collateral circulation via arterial arcades. Venous drainage parallels the arterial supply and drains ultimately into the superior mesenteric vein which joins the splenic vein to form the portal vein.

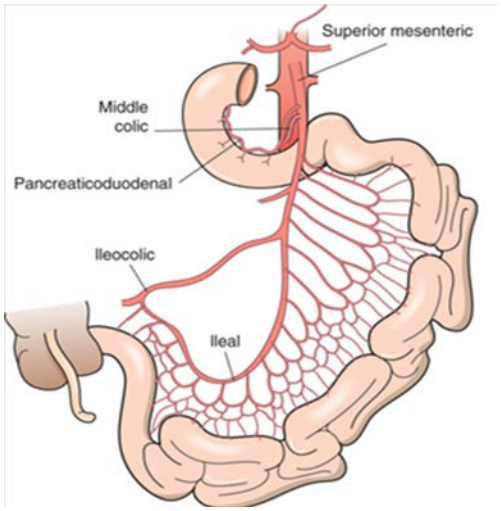


Fig. 1 Blood Supply of Small Intestine

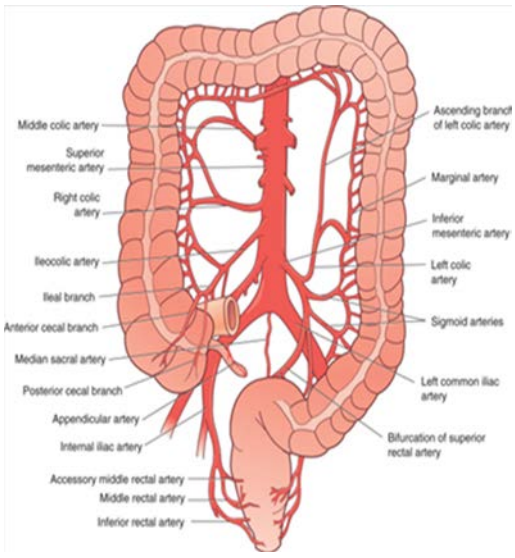


Fig. 2 Blood Supply of Large Intestine

Caecum upto the proximal transverse colon are supplied by the ileocolic and the right colic arteries, branches of the superior mesenteric artery. Middle colic artery again a branch of the SMA supplies the proximal and distal transverse colon. Inferior mesenteric artery supplies the descending colon, sigmoid colon and the upper part of the rectum

through the left colic artery, sigmoid branches and the superior rectal artery. At the region of the splenic flexure, there may be an overlap of supply between the territories of the middle colic and the left colic arteries. This forms the most inconsistent of the collateral formation in the entire colon termed as the watershed area of the colon. Hence anastomosis is generally avoided in this region for the fear of anastomotic leaks owing to poor vascular cover. The arc of Riolan, also known as the meandering mesenteric artery is an important collateral vessel connecting the two major vascular pedicles of the intestine namely the superior and inferior mesenteric pedicles. Venous drainage is to the inferior mesenteric vein that empties into the splenic vein.

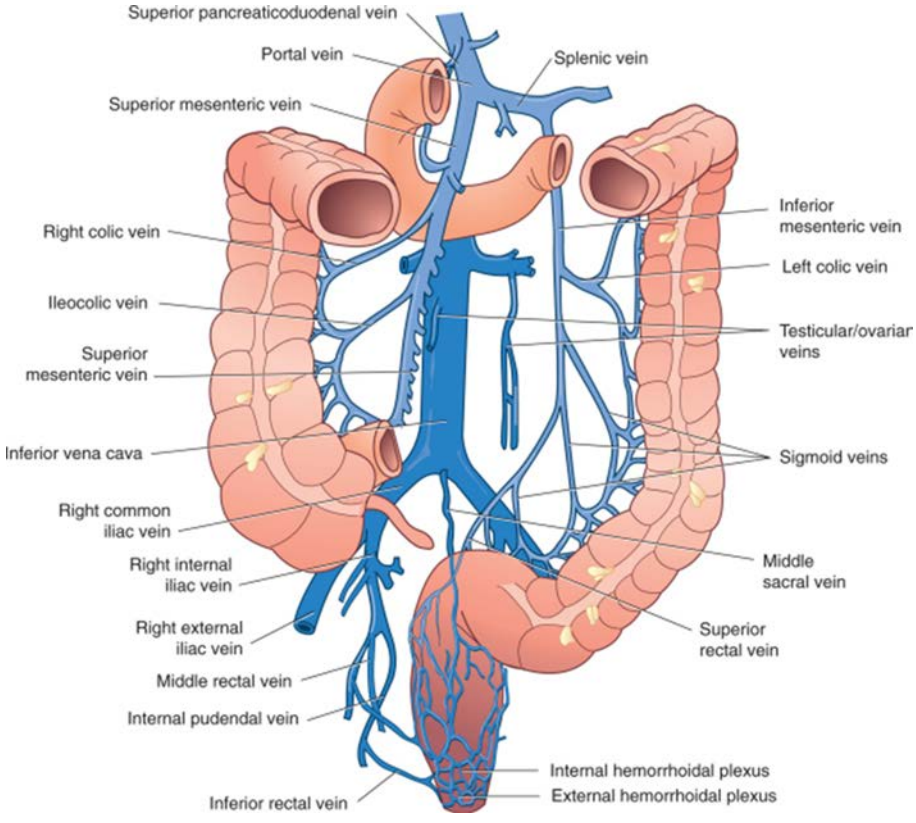


Fig. 3 – Venous Drainage of Intestines

Lymphatic drainage:

Lymphatic drainage of the small intestine proceeds from the mucosa through the wall to the adjacent nodes in the mesentery onto regional nodes in the arterial arcades finally to the nodes around the base of the superior mesenteric artery.

Lymphatics from the colon and proximal two thirds of the rectum ultimately drain into the para-aortic nodal chain, which empties into the cisterna chyli. Lymphatics draining the distal rectum and anal canal may drain either to the para-aortic nodes or laterally, through the internal iliac system, to the superficial inguinal nodal basin.

INNERVATION [1, 2, 3]

Innervation of the small bowel is provided by both parasympathetic and sympathetic systems. Vagus, via the coeliac ganglion supplies the parasympathetic component affecting almost all phases of intestinal activity including secretion and motility. Sympathetic fibres arising from three sets of splanchnic nerves supply the intestines through plexus around the arterial arcades. Pain is mediated through general visceral afferent fibres in the sympathetic system. In general, parasympathetic activity increases the activity of intestinal smooth muscle and sympathetic system generally decreases the smooth muscle activity while causing the sphincters to contract.

Innervation of the large bowel is by a complex plexus of fibres. Sympathetic fibres are derived from the T6 to T12 preganglionic fibres and the lumbar sympathetic system. Parasympathetic system is by the vagus on the right side and the pelvic parasympathetic fibres or the nerve erigenti on the left side.

The intestines also have a set of intrinsic nervous system called the enteric nervous system. This is comprised of

1. Auerbach's Myenteric plexus between outer longitudinal and middle circular fibres. This supplies the smooth muscular layers and is primarily concerned with motility of the intestines

2. The Meissner’s Submucus plexus found between the middle circular layer and the mucosa. This innervates the glandular epithelium, endocrine cells and the submucosal blood vessels. It is primarily involved with intestinal secretion.

   The various neurotransmitter peptides associated with the enteric nervous system are:

   1. CGRP
   2. CCK
   3. Endothelin-2
   4. Enkephalins
   5. Galanin
   6. GRP
   7. Neuropeptide Y
   8. Neurotensin
   9. Peptide YY
   10. PACAP
   11. Somatostatin
   12. Substance
   13. PTRH
   14. VIP

MICROSCOPIC ANATOMY [1, 2, 5, 6]

The bowel wall consists of four layers from without inwards namely-

1. Serosa
2. Muscularis Propria made up of a thin outer longitudinal layer and a thicker inner circular layer.
3. Submucosa made of fibroelastic connective tissue and is the strongest layer of the bowel wall
4. Mucosa with three components - muscularis mucosa, lamina propria, and epithelial layers.

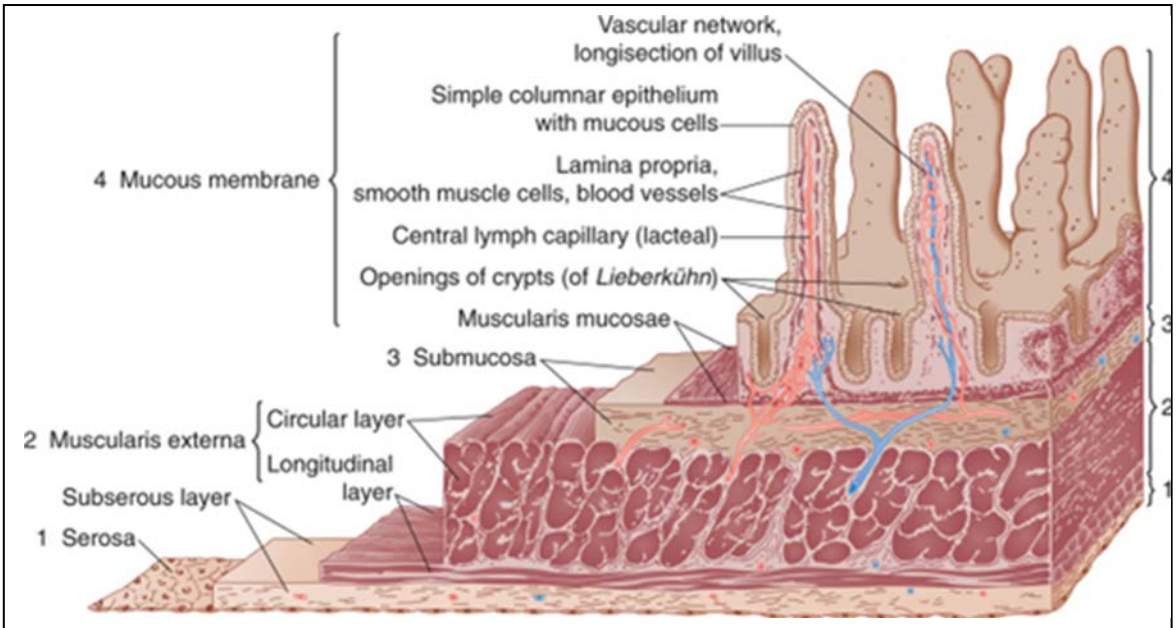


Fig. 4 Microscopic Anatomy of small bowel wall

The entire mucosa is arranged in the form of villi which increases the effective absorptive surface area of the intestines. These are again made up of microvilli which further increase the absorptive area by 30 fold. Mucosa is further made of four major cell types namely-

1. Goblet cells, which secrete mucus
2. Paneth cells, which secrete lysozyme, tumor necrosis factor (TNF), and the cryptidins and thought to be related to the host mucosal defence system
3. Absorptive enterocytes
4. Enteroendocrine cells, of which there are more than 10 distinct populations that produce the gastrointestinal hormones

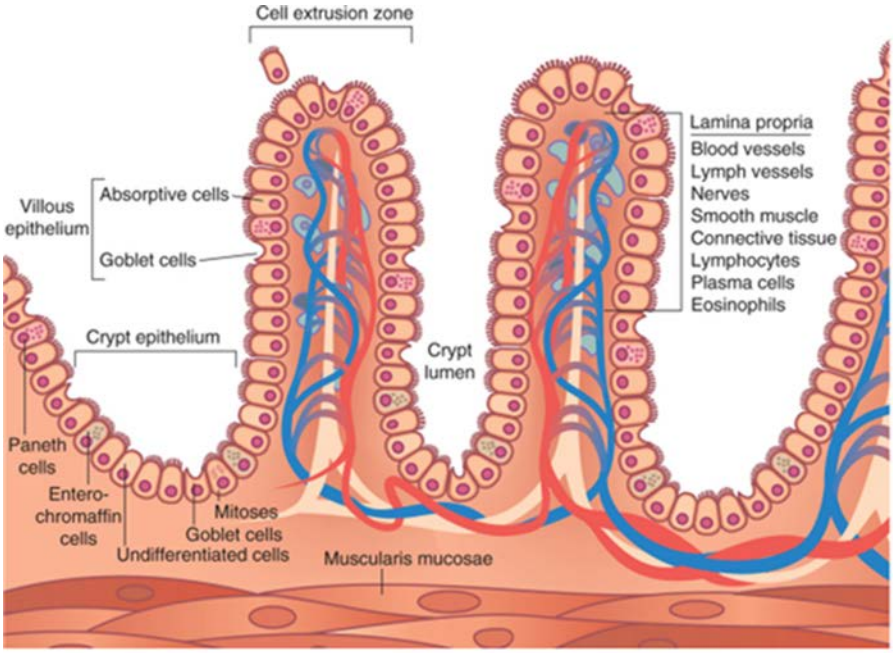


Fig 5 Microscopic anatomy of Villi

PHYSIOLOGY [3, 5, 9]

One of the most important organ systems of the body, the intestines play an important role in many of the body functions

1. Immunity & Barrier function
2. Digestion & absorption of nutrients
3. Fluid and electrolyte balance
4. Motility and propulsion

A distinctive feature of the intestinal epithelial system is the ability to seal the luminal contents off from the intracellular environment. This is achieved by numerous surface proteins namely occludins and claudins which influence the selective permeability and tight junctions. Loss of this effective barrier system allows free movement of substances across the barrier resulting in major fluid electrolyte disturbances and translocation of bacteria from lumen into the bowel wall.

The human GIT is the single largest organ of immune system in the body. It is capable of modifying both the local and systemic immune responses. Local immune activity is against various intraluminal toxins and pathogens. Systemic modulation is by balancing mucosal defense and potential overstimulation of the immune system. Secretory immunoglobulins help in neutralizing luminal antigens. The epithelial layers also play an important role in antigen presentation and providing

important immune regulatory information to the underlying lymphocytes. Gut function is also affected in response to the alterations in the immune system. Food allergies and inflammatory bowel disease may cause increased fluid secretion and diarrhea.

Various fluids are secreted by the GIT as shown in the table below.

Ingested fluids	2000
Endogenous secretions 1. Saliva – 1500 2. Stomach – 2500 3. Bile – 500 4. Pancreas – 1500 5. Intestines – 1000	7000
Total input	9000
Absorption 1. Jejunum – 5500 2. Ileum – 2000 3. Colon – 1300	8800
Net fluid remaining in stools	200

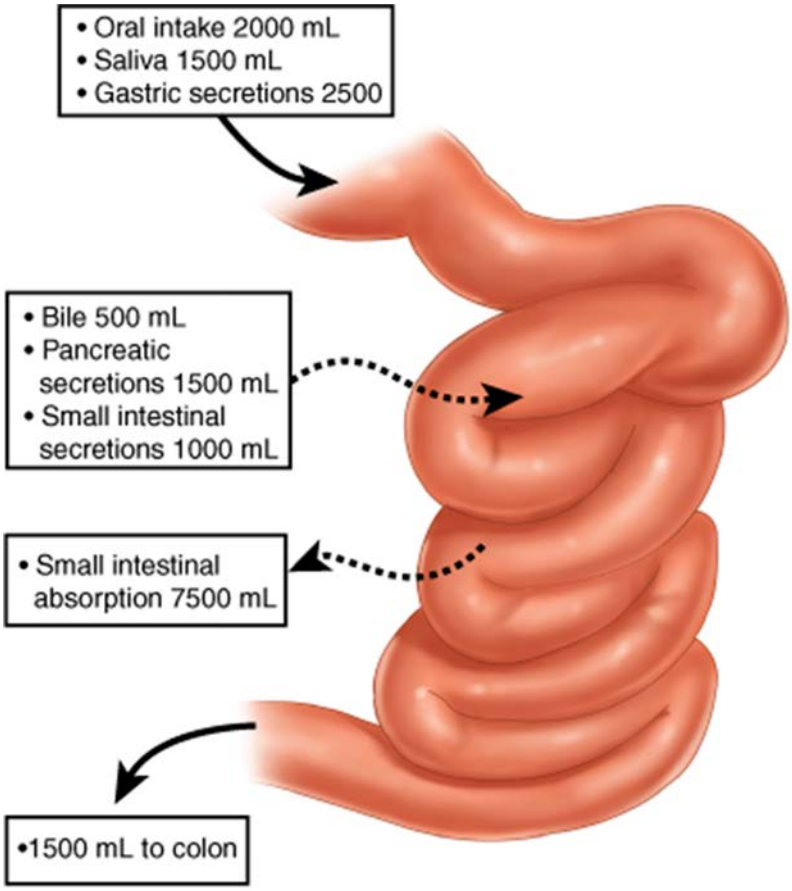


Fig. 6 Fluid absorption in Small Intestine

Majority of the fluid that enters the intestine is absorbed. Hence whenever there is a failure of absorption or increased secretion, the intestines get distended as in case of obstruction.

Motility of the gut acts as a major contributory aid in digestion. There are basically three patterns of contraction –

a. Antegrade propulsive movements that propel the contents forward at a rate of 1-2 cm per minute and
b. Segmentation contractions causing to and fro movement of food that increases mixing the food with the digestive enzymes and bringing it in contact with the mucosa
c. Tonic Contractions that help in isolating one segment of intestine from the other

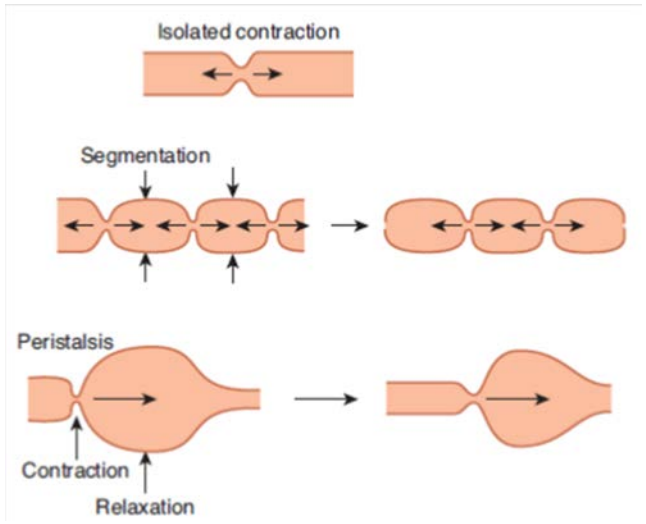


Fig. 7 Types of Peristalsis

The interstitial cells of Cajal have been identified as the generator of pacemaker potentials. Spontaneous depolarizations of the intestinal smooth muscles are also observed. The entire smooth muscle architecture

of the bowel acts as a syncytium and action potentials can travel through the gap junctions thus resulting in a coordinated muscle contraction.

Digestion and absorption is the primary function of the GIT. With the help of gut motility and various digestive fluids and enzymes, the ingested complex food materials are broken down into their basic molecules which are then absorbed through various membrane transporters in the brush border epithelium actively. The remaining undigested and unabsorbed food is propelled out as fecal matter.

INTESTINAL OBSTRUCTION

Definition & Classification

Bowel is said to have been obstructed when the normal passage of Intestinal contents does not occur [7]. Intestinal Obstruction can be classified into various types as follows [9]

Based on Anatomic Segment involved

1. Small Bowel Obstruction
2. Large Bowel Obstruction
3. Generalized Obstruction involving both Large & Small Bowels

Based on the level of Obstruction

1. Proximal – Pylorus, Duodenum , proximal Jejunum
2. Intermediate – Mid jejunum to mid ileum
3. Distal – Distal Ileum, Ileo-caecal valve, Proximal colon
4. Most distant or Low Obstruction – Beyond transverse colon

Based on Etio-pathogenesis

1. Dynamic or Mechanical where peristalsis is working against a mechanical block
2. Adynamic or Functional where there is no peristalsis

Based on Time since obstruction

1. Acute – hours to 1 day
2. Sub-acute – days to week
3. Chronic – Unrelenting obstruction lasting several weeks

Based on Extent of Obstruction

1. Complete
2. Partial

Based on the type of Obstruction

1. Simple
2. Complicated where there is compromise of bowel vascularity as in Closed loop obstruction & Strangulation

Natural History [5, 7, 9, 10]

Irrespective of the type or category of Intestinal obstruction, the natural history of the disease follows somewhat a fixed course. The segment of intestine proximal to the site of obstruction develops distension and altered mobility. The distension is mainly caused by two factors.

1. Gas – Early phase due to swallowed air. Later phase due to fermentation of intra-luminal contents due to the significant overgrowth of bacteria.
2. Fluid – mainly composed of digestive juices and mucosal secretion in the background of reduced absorption. Sequestration of fluid combined with repeated vomiting causing massive changes in fluid-electrolyte homeostasis leading to dehydration, hypovolemia and shock.

The segment distal to the obstruction continues to function normally, absorbing and propelling its contents until it becomes empty. At this stage, the bowel becomes contracted and immobile. In the early period, peristalsis in the proximal bowel increases both in number and intensity in an attempt to overcome the obstruction. This increased activity is directly proportional to the distance of loop from the site of obstruction. If the bowel obstruction is unrelieved at this stage, the proximal loop

begins to dilate, thereby reducing the efficacy of peristalsis and finally becomes flaccid. This flaccidity is a protective mechanism so as to prevent compromise in vascularity that occurs in response to the persistently raised intraluminal pressure. With progressive distension, the intra-mural pressure increases initially to hamper the venous outflow. This further raises the intramural pressure above the intra-arterial pressure thereby decreasing blood supply to the segment. Subsequently if left untreated, the intestine goes in for ischemia, hemorrhagic infarction and perforation which cause peritonitis culminating ultimately in shock and death of the individual.

PATHOGENESIS [7, 9, 10]

Alterations in Fluid electrolyte balance

Normal intestines have a tremendous capacity of absorption of fluids and electrolytes. With any form of obstruction, fluid and electrolytes start to accumulate inside the lumen distending the lumen. In the first 12 hours of disease, this accumulation is due to reduced absorption. By 24 hours, the accumulation is more rapid due to further reduction in absorption along with commensurate increase in secretion. Prostaglandin synthesis is enhanced in response to bowel wall stretching. Prostaglandins are known to enhance intestinal secretion. However the exact pathogenetic mechanism has not yet been delineated. Anti-inflammatory agents have been demonstrated to reduce intestinal secretions. Further inflammation has been demonstrated proximal to the site of obstruction. Hence inflammatory mediators are suspected in increasing the intraluminal fluid secretion. Enteric nervous system largely controls the mucosal absorption and secretion. Release of bioactive peptides through their vasoactive as well as neuroactive properties have been implicated in the increased intraluminal fluid accumulation. The iso-osmolar sequestration results in a net iso-osmolar volume contraction. This causes a major disruption of the fluid and electrolyte homeostasis. This disruption is further complicated by recurrent vomiting.

Alterations in Gut Motility

Whenever there is a partial obstruction of the bowel, the proximal bowel contracts vigorously to get the contents past the site of obstruction. However, if there is a total occlusion, the proximal bowel gradually dilates and eventually initiates retrograde giant contractions as in the first phase of vomiting. These changes are thought to be mediated via nitrous oxide, VIP and few other neuro-humoral pathways yet to be clearly elucidated.

Alterations in gut Microbiology

Jejunum and proximal ileum are virtually sterile despite the existence of resident flora at a low concentration. These may translocate to infect the mesenteric nodes and systemic organs in the absence of a fully functional mucosal barrier which often is the case in later phases of obstruction. This phenomenon justifies the use of prophylactic antibiotics in all cases of mechanical intestinal obstruction.

Alterations in blood flow

In the initial stages of obstruction and bowel dilatation, intraluminal pressures remain below 8mm Hg, which rapidly peaks as closed loop obstruction sets in. Experimental models have demonstrated

that increase in luminal pressures to as low as 15mm Hg causes substantial reduction in mucosal blood flow, and at pressures of 20mm Hg, there occurs shunting of blood flow from mucosa to the outer wall layers. This leads to hypoxia and relative ischemia to the villous tips, and free oxygen radical production and release of endothelins. The further course of events finally leads to the disruption of barriers between luminal endotoxin pool and the circulation, reducing the chances viability of bowel and survival of the individual.

Systemic and hemodynamic alterations

Isotonic volume contraction initially occurs in bowel obstruction secondary to intestinal and peritoneal sequestration of extracellular fluid and losses due to vomiting. Renin-Aldosterone mechanism is activated in response to the hypovolemia. This combined with persistent vomiting and intestinal sequestration lead to hypokalemia and its sequellae. Cardiac manifestation occurs as tachycardia and increased cardiac irritability. Respiratory compromise occurs due to abdominal distension and aspiration pneumonitis. Neglected obstruction further leads to pre-renal azotemia and acute renal failure. To summarize, untreated intestinal obstruction leads to multi organ dysfunction.

Etiology of Mechanical Obstruction of Bowel [9]

Extramural	Intramural	Intraluminal
A. Adhesions a. Congenital b. Inflammatory c. postoperative B. Herniae a. External b. Internal C. Congenital a. Annular Pancreas b. Volvulus c. Peritoneal Encapsulation D. Neoplastic a. Carcinomatosis b. Extraintestinal E. Inflammatory a. Intraabdominal Abscess b. Peritonitis c. Splenosis F. Miscellaneous a. Superior Mesenteric Artery syndrome	A. Congenital a. Malrotation b. Meckel's Diverticulum c. Diverticulum & Cysts B. Inflammatory a. Infections b. Inflammatory Bowel Disease c. Eosinophilic Granuloma C. Neoplastic a. Primary b. Secondary D. Traumatic a. Hematoma b. Ischemic Stricture E. Miscellaneous a. Intussusception b. Endometriosis c. Strictures	A. Gall Stone Ileus B. Enterolith C. Bezoar D. Foreign Body E. Parasite F. Cholestyramine G. Intraluminal Diverticulum H. Intraluminal Polyp

STRANGULATING OBSTRUCTION [4]

In this type of obstruction, the viability of the intestines is threatened secondary to compromised blood supply. Various causes of strangulation are:

1. External	a. Hernia b. Adhesions/ Bands
2. Interrupted blood flow	a. Volvulus b. Intussusception
3. Increased intraluminal pressure	a. Closed loop Obstruction
4. Primary	a. Mesenteric infarction

Once blood supply is compromised, hemorrhagic infarct supervenes followed by systemic exposure to bacteria and toxins. Mortality of intraperitoneal strangulation is greater than external herniae as segment involved is greater, and resultant fluid electrolyte imbalance is greater. Closed loop obstruction occurs when bowel is obstructed at both proximal and distal ends. Distension of proximal bowel occurs late when imminent gangrene of strangulated segment causes retrograde thrombosis of mesenteric veins. A classic example of closed loop obstruction is malignant stricture of right colon with a competent ileo-caecal valve and volvulus.

PRESENTATION [4, 7, 9, 10]

Nausea & Vomiting, colicky abdominal pain, Obstipation and abdominal distension are the four cardinal features of intestinal obstruction. However the order in which these features appear depends on

1. The location of the obstruction
2. The age of the obstruction
3. The underlying pathology
4. The presence or absence of intestinal ischaemia

Proximal obstruction may present with only nausea and vomiting.

PAIN

The most prominent symptom with distal obstructions is episodic colicky abdominal pain. Pain may be however absent if the bowel is able to decompress fully into the stomach. The classical description of the pain is crescendo-decrescendo type of pain. [4]. Pain lasts for 1-3 minutes and in between the episodes, patient may be pain free in the absence of complications.

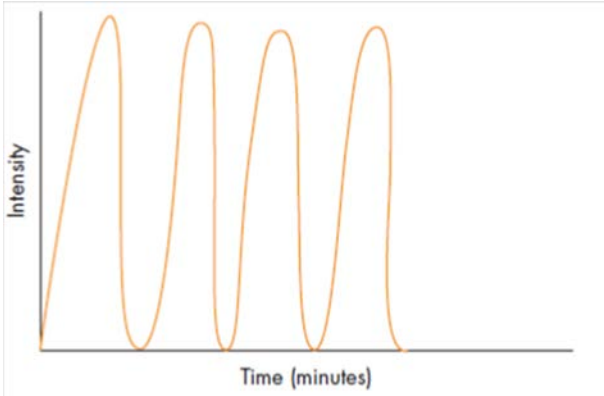


Fig 8 Graphical representation of pain in obstruction

The quiescent interval may be 1-3 minutes in proximal obstructions and 10-15 min in more distal obstructions. Pain is often synchronous with borborygmi. Patients are often restless in search of a comforting position. This is in contrast to patients of peritonitis where they lie still to avoid any movement that causes pain.

VOMITING

Vomiting occurs early in proximal obstruction, but late or may even be conspicuously absent in distal obstruction. Relentless vomiting may be the initial presentation in early closed loop obstruction. Initial vomitus contains altered food contents, progressing to bilious fluid and in late obstruction it may turn feculent with a foul smelling odour, owing to the increased proliferation of bacteria and alteration of the luminal contents and pH by the luminal bacteria.

CONSTIPATION

Obstipation is a late event in most obstructions. Patients with partial obstruction may continue to pass flatus and have explosive bouts of diarrhea with relief of pain. Even in complete obstruction, patient may continue to have bowel movements owing to the increased peristalsis in the bowel distal to obstruction. As the unobstructed bowel empties, obstipation gradually supervenes. As the obstructed intestine dilates, the patient gradually develops abdominal distension. This is more prominent

in mid gut and distal obstruction and less common in more proximal obstruction. Constipation may be absent in certain types of obstruction as in-

1. Richter's hernia
2. Mesenteric vascular occlusion
3. Gallstone ileus
4. Obstruction associated with pelvic abscess
5. Partial obstruction (faecal impaction/colonic neoplasm) in which diarrhoea may often occur.

On examination, one may appreciate signs of dehydration such as sunken eyes, dry mucous membranes, loss of skin turgor. Tachycardia and hypotension may indicate severe dehydration, peritonitis, or both. In late presentations, patient may have a toxic look secondary to septicaemia. Fever often suggests the possibility of strangulation or some other complication of the underlying process associated with peritonitis.

Fever may also suggest the underlying etiology as in inflammatory conditions as diverticulitis, inflammatory bowel disease and localized perforation.

Abdomen must be inspected for any evidence of scars of previous surgeries and all scars must be accounted for, as it may support a suspicion of adhesive obstruction. Abdomen is often distended with visible intestinal peristalsis, especially in thin individuals. Gross distension is often appreciated in cases of sigmoid volvulus and other late presentations.

Fig 9 Gross abdominal distension

Fig. 10 Feculent Ryles tube aspirate in late intestinal obstruction

Fig 11 Visible intestinal peristalsis

Mild generalised tenderness is almost universally present. Frank signs of peritonitis such as localized tenderness, guarding and rigidity

should alert to the possibility of complication of obstruction such as a strangulating obstruction or an alternative diagnosis. Etiological features such as presence of mass in the abdomen and an external hernia must be noted on a thorough examination.

Percussion often reveals tympanic note due to gaseous distension of bowel loops. In also helps identify free fluid in peritoneal cavity and detect rebound tenderness in case of peritonitis.

Auscultation reveals increased bowel sounds separated by periods of silence initially. As the bowel gradually loses its contractility, a silent abdomen prevails making distinction from adynamic ileus difficult. This is an ominous sign as it may mark bowel fatigue and impending complication. Gastric succussion splash in a patient who has not consumed orally in the last two hours is often a strong evidence of gastrointestinal obstruction. Digital rectal examination is mandatory. It may reveal faecal impaction, palpable growth or mass as a cause of the obstruction. Presence or absence of faecal staining must be noted and any altered staining also observed such as hematochezia which may suggest a mucosal lesion, as in strangulating lesion, cancers or intussusception.

Ryle’s tube aspirate may be bilious in proximal obstructions and feculent in case of late presentation of proximal obstruction and distal obstruction.

INVESTIGATIONS [4, 5, 9, 10, 11]

Routine baseline laboratory investigations, though not of much use in the diagnosis of intestinal obstruction, are mandatory to assess the hemodynamic status and to guide resuscitation. These include a baseline Hemogram, electrolyte and acid base balance assessment and assessment of any comorbidity. Various parameters such as raised leucocytes, acidosis, serum phosphate, Creatinine phosphokinase, intestinal fatty acid binding protein have been assessed in the predicting of vascular compromise of the gut. However, none of them are specific or sensitive enough to draw definitive conclusions. Leukocytosis occurs usually once the effects of the obstruction are systemic as occurs when either peritonitis or sepsis sets in. Secondary metabolic alterations can manifest in the form of acidosis and elevated renal parameters. The parameters such as persistent pain, fever, leukocytosis, tachycardia and features of peritonitis as guarding and abdominal rigidity have been evaluated to predict the risk of strangulation preoperatively. Some studies have shown that presence of abnormality in three or more of these parameters have a positive predictive value of over 80% to predict strangulating obstruction, which approaches 100% positive predictive value when there are abnormalities in four or more of the above parameters.

IMAGING [9, 10, 11]

Radiological investigations are primarily used to confirm the diagnosis of intestinal obstruction and to exclude other pathologies. Sometimes it may also help to find the site of obstruction and even the causing factor.

Plain abdominal radiography still remains the initial investigation modality in all patients with suspected obstruction. Of late, the diagnosis rests on a supine film, with erect films requested for when further doubt exists. Plain X-rays show dilated bowel loops filled with gas and fluid, often layering out in a stepwise pattern with multiple air-fluid levels. Fluid levels appear later than gas shadows, as it takes time for the air and fluid to separate. Small bowel loops more than 3cm diameter and proximal colon more than 8-15cm in diameter and distal colon more than 4cm are regarded to be dilated.

Various features of intestinal obstruction on radiography are-

1. The obstructed small bowel is characterised by straight segments that are generally central and lie transversely. No gas is seen in the colon
2. The jejunum is characterised by its Valvulae conniventes, which completely pass across the width of the bowel and are regularly spaced, giving a ‘concertina’ or ladder effect

3. Ileum is classically identified as a featureless loop
4. Caecum – a distended caecum is shown by a rounded gas shadow in the right iliac fossa
5. Large bowel, except for the caecum, shows haustral folds, which, are spaced irregularly, do not cross the whole diameter of the bowel and do not have indentations placed opposite one another

Small bowel dilation occupies the center and large bowel loops, the periphery of the abdominal radiograph. Isolated dilation of large bowel with undilated small bowel signifies a large bowel obstruction with competent ileo-caecal valve, an example of closed loop obstruction which is regarded a surgical emergency. Free intra-peritoneal air suggests a complication as perforation of a hollow viscus. Intraluminal foreign bodies may be visualized and air in the biliary tree suggests biliary-enteric fistula and is indicative of gall stone ileus.

Contrast Radiographs are often helpful to localize the site of pathology and also sometimes to find the nature of the underlying lesion. This may be done from above as a small bowel follow through or enteroclysis or from below as a contrast enema as done for large bowel pathologies. They are especially helpful when the diagnosis is in doubt as in early closed loop obstruction.

Certain signs have been identified in some specific conditions as-

1. Step ladder appearance – small bowel obstruction
2. Coffee bean sign and bird's beak sign – sigmoid volvulus
3. Claw sign – intussusception

Ultrasonography aids in the diagnosis of intestinal obstruction in both the etiology and location. Unlike X-rays or CT scans, there is no risk of radiation exposure and also ultrasonography provides real time imaging. Examination of blood flow by Doppler flowmetry improvises the diagnostic accuracy of vascular compromise. However it is highly operator dependent and less yielding in emergency situations cases of gaseous distension due to acoustic shadowing.

Computed tomography has evolved as an indispensible radiological tool in imaging the intestines. Whereas X-rays and contrast studies are able to image the luminal surface only, Computed tomography can pick up intra-luminal, intramural and also extra-mural pathologic entities. Thereby it offers to find out the precise site by clearly demarcating the transition zone from obstructed to the non-obstructed bowel, as well as the cause of the obstruction in most cases especially where diagnosis is in doubt. Delayed or absent enhancement of bowel wall on IV contrast studies improves the preoperative predictive accuracy of strangulating

obstruction and ischemia, thereby significantly improving the diagnostic efficiency.

Colonoscopy often reveals the underlying pathology in intraluminal causes of large bowel obstruction as in fecal impaction and malignant growths, diverticulitis, etc.

Recent advances in imaging modalities include;

1. Capsule endoscopy [4]
    1. Advantages – painless & able to visualize entire small bowel
    2. Disadvantages – no accurate localization, incomplete studies due to battery life, cannot be used in complete obstruction
2. Chromo-endoscopy [4, 5]
3. Virtual endoscopy and virtual colonoscopy

These may help identify many intraluminal as well as mural diseases.

However, when the clinical suspicion suggests a strangulating obstruction, time must not be lost in a complex battery of investigations as such studies may not necessarily alter management plans. The information obtained from an investigation must be weighed against the time lost and the delay of surgical intervention. [9]

Fig. 12 Plain X-ray Erect Abdomen

Step Ladder Pattern

Fig. 13 Plain X-ray Erect Abdomen

Multiple Air-fluid levels

Fig. 14 Barium Enema – Obstruction in Ascending Colon

Fig 15 Contrast enema - Claw sign - Intussusception

Management [4, 5, 6, 7, 8, 9, 10]

Intestinal obstruction is to be diagnosed and treated at the earliest due to the concern for strangulating obstruction and in view of the complications it can cause. The bowel in strangulating obstruction can go in for gangrenous changes within 6 hours. A common saying is that ‘the sun must not both rise and set on a case of unrelieved intestinal obstruction’. Early diagnosis and prompt intervention are mandatory to reduce the risk of vascular compromise of the bowel and hence reduce the morbidity and mortality of intestinal obstruction.

Initial management consists of correction of fluid and electrolyte imbalances. Vigorous hydration is done with isotonic sodium chloride solution with constant monitoring of central venous pressure, pulmonary arterial pressure, urine output, and where indicated, arterial blood gas values. Once urine output has stabilized, potassium must be added to the resuscitation protocol as persistent vomiting significantly depletes body potassium stores.

Nasogastric decompression of stomach is essential to reduce the risk of aspiration and minimize further intestinal distension by swallowed air. It also helps in relief of symptoms of distension and helps improve the ventilatory status in patients with respiratory compromise. However a nasogastric tube is often ineffective for decompression of intestines distal

to ligament of Trietz [9]. Use of longer nasogastric tubes had been advocated in situations where an early operation is not feasible as in patients with partial obstruction arising from Crohn's disease, peritoneal carcinomatosis, radiation enteropathy, or many previous laparotomies for obstruction.

A brief period of non-operative management may be made in carefully selected cases with termination of expectant management should the patient fail to improve. Nonoperative management is always undertaken with a calculated risk for overlooking an underlying strangulation obstruction. The mortality rate from obstruction with irreversible ischemia (gangrene) ranges from 5 to 31%, whereas with simple mechanical obstruction relieved within 24 hours, the mortality rate is about 1% [9]. This reinforces the importance of constant monitoring, and as no test reasonable predicts with certainty about strangulating obstruction, surgical intervention must be planned at the earliest possible time soon after adequate resuscitation. De-novo obstruction of the small bowel and obstruction secondary to intra-abdominal malignancies often do not respond well to non-operative management and early surgery is indicated.

The timing of operation depends primarily on three factors [9]:

1. Duration of obstruction, that is, severity of fluid, electrolyte, and acid-base abnormalities
2. Opportunity to improve vital organ function
3. Consideration of the risk for strangulation

The indications of surgery in a patient with possibility of intestinal obstruction are [4, 9] –

1. Acute small bowel obstruction
2. Persistent Rapidly progressing constant, noncrampy abdominal pain or distension, with or without features of peritonitis
3. Development of peritoneal findings, fever, tachycardia, diminished urinary output, leucocytosis, hyperamylasemia, and metabolic acidosis
4. Failure of an obstructive picture of complete obstruction to resolve within 12 to 24 hours, even in the absence of evolving symptoms of peritoneal findings
5. Large bowel obstruction and acute onset of small bowel obstruction

Once a decision for operative management has been made, patient should be given prophylactic, broad-spectrum antibiotics covering gram-negative aerobes and anaerobes (particularly if strangulation is suspected) to minimize infective complications if resection proves necessary or peritoneal spillage occurs as the result of an inadverant enterotomy. However in patients put on conservative management with observation alone, antibiotics are controversial and are of questionable value and moreover they can obscure an underlying process delaying optimal therapy [9]. At operation all effort must be made to accurately identify the underlying cause and correct the obstruction. Assessment of bowel viability is of paramount importance as described below [4].

Intestine	Viable	Non-viable
Circulation	Dark colour become lighter; Mesentery bleeds if pricked	Dark colour remains, no bleeding of mesentery if pricked
Peritoneum	Shiny	Dull and lustreless
Intestinal musculature	Firm, pressure rings may or may not disappear, Peristalsis may be observed.	Flabby, thin and friable. Pressure rings may persist, No peristalsis.

Objective evidence of intestinal viability is provided by fluorescein dye studies. Doubtfully viable bowel must be observed after packing with warm pads and observing for changes to normal colour and peristalsis

SPECIFIC TYPES OF OBSTRUCTION

ADHESIVE OBSTRUCTION [4, 5, 9, 10]

In view of the common occurrence of intra-abdominal surgeries, adhesive obstruction has become the most common cause of mechanical intestinal obstruction especially in the west. This may be particularly difficult to differentiate from paralytic ileus in the early postoperative period. Any source of peritoneal irritation results in local fibrin production, which produces adhesions between apposed surfaces. Early fibrinous adhesions may disappear when the cause is removed or they may become vascularised and be replaced by mature fibrous tissue. Some of the common causes of adhesions are [4]:

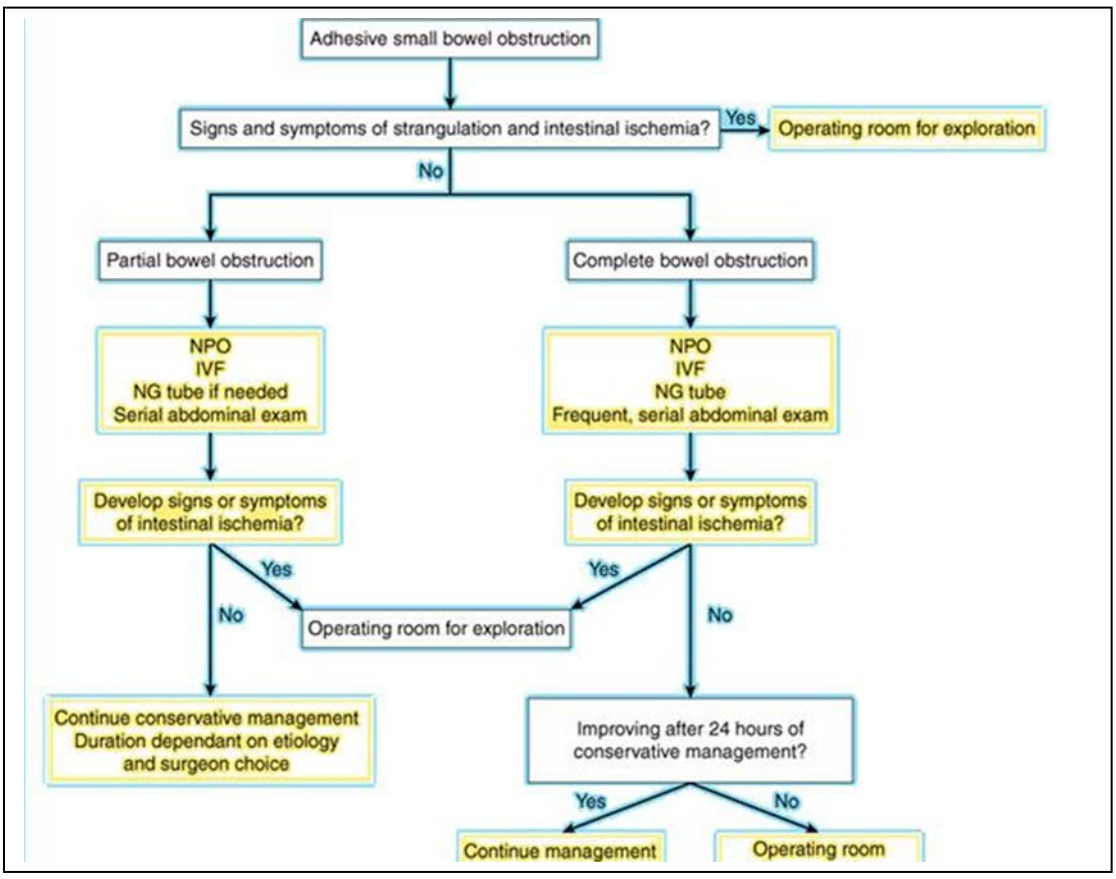
a. Ischaemic areas as in Sites of anastomoses, reperitonealisation of raw areas, trauma, vascular occlusion
b. Foreign material such as Talc, starch, gauze, silk in the peritoneal cavity
c. Infection as in Peritonitis, tuberculosis
d. Inflammatory conditions such as Crohn's disease

Adhesions are classified into various types [9] based on etiology as congenital, postoperative, inflammatory etc., based on duration as early fibrinous and late fibrous adhesions. However, from a practical perspective, they are classified into easy flimsy adhesions, and late dense

adhesions. Postoperative adhesions giving rise to intestinal obstruction usually involve the lower small bowel. Operations for appendicitis, lower abdominal surgeries and gynaecological procedures are the most common precursors and are an indication for early intervention [9]. Many substances such as steroids, anti-inflammatory substances, and hyaluronidase have been instilled into the peritoneal cavity to prevent adhesions, but so far, none have been found to be particularly useful.

Bands are another common cause of obstructions. These may be divided as –

a. Congenital – obliterated vitellointestinal duct
b. String bands following peritonitis or other inflammatory pathology
c. Bands of omentum adherent to the parietes.



The initial line of management recommended in most patients of adhesive intestinal obstruction is conservative line, which succeeds in more than 75% of cases. The remainder however require surgical intervention to release the adhesion/band. Only the culpable band / adhesion needs to be released as releasing all adhesions only further aggravates the problem. Various definitive surgical options in recurrent adhesions are [4]-

1. Noble's plication of intestines
2. Child-phillips mesenteric plication
3. Intestinal intubation using Intraluminal splinting

Fig. 17 Multiple Interbowel loop adhesions causing obstruction

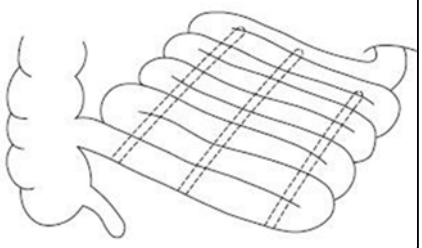


Fig. 18 Charles Phillips procedure

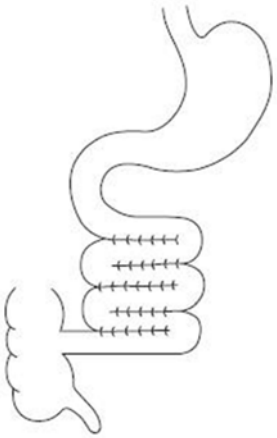


Fig. 19 Noble's plication

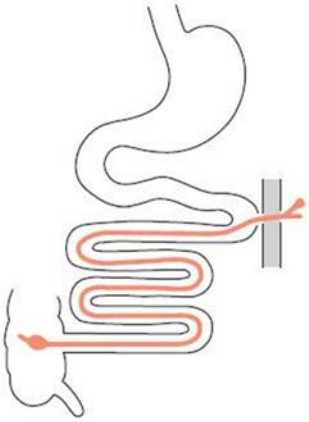


Fig. 20 Baker's tube insertion via Witzel Jejunostomy

None of them have established their superiority over the other methods in the management. Prevention is better than cure. Methods that can be adopted to prevent / reduce adhesion formation are [4, 9]-

1. Good surgical technique with meticulous hemostasis
2. Washing the peritoneal cavity with saline to remove clots, debris & other foreign material
3. Minimising contact with gauze pieces
4. Covering of anastomotic sites and other raw surfaces
5. Bio-absorbable membrane barriers to prevent contact of vulnerable surfaces

HERNIAE [4, 5, 6, 7, 8, 9, 10]

They are the second most common cause of intestinal obstruction. Internal herniae are detected only at laparotomy. Almost all hernias with evidence of obstruction, even if only partial, need emergency surgical intervention due to the high risk of strangulation.

Femoral and umbilical hernias are at a higher risk of obstruction due to the narrow neck [4, 8], though inguinal hernia accounts for more number of cases due to the common incidence.

The constricting agent causing the obstruction is mostly the neck of the sack (deep ring in inguinal hernias, ileopectineal ligament in femoral hernia and the fascial defect as in umbilical/ other ventral hernia) followed in frequency by adhesions within the sac of the hernia [4]. Irrespective of the type of hernia, an obstructed or strangulated hernia is almost always symptomatic and mandates emergent intervention in order to avoid complications. Certain types of hernias such as Richter's hernia may have strangulation of partial bowel wall without complete obstruction.

The treatment of choice is emergency exploration. At exploration the hernia needs to be opened from the neck in non-strangulating hernias. In case of suspicion of strangulation, sac is to be opened from the fundus and toxic fluid if any must be let out to avoid peritoneal contamination. Adhesions if any are to be gently released, viability of the bowel

assessed. If the bowel is viable, the constricting ring is released and the contents reduced back to the abdomen. In nonviable bowel, a resection of gangrenous segment is tone with primary anastomosis. The hernial orifice must be meticulously repaired to avoid recurrence. Use of prosthetic mesh in cases where the incision has been soiled or where gangrenous bowel has been resected is associated with a high risk of infection. Biosynthetic meshes made of collagen and dermis are more suited to be used in such situations [4].

Fig 21 Strangulated Incisional Hernia

Fig. 22 Strangulated Inguinal Hernia

Fig 23 Obstructed Umbilical Hernia

Fig 24 Strangulated Femoral Hernia

Fig 25 Strangulated inguinal hernia with non-viable segment

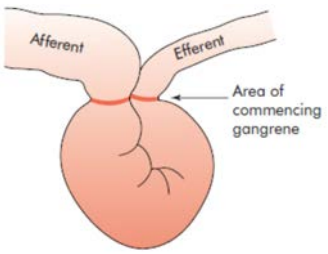


Fig 26 Area of commencement of gangrene in strangulation

INFLAMMATORY CAUSES OF OBSTRUCTION [4, 7, 9]

1. Diverticular disease

Diverticulosis is actually an extraluminal pericolic infection caused by the extravasation of feces through the perforated diverticulum which has been controlled by the body's natural defences.

Sigmoid colon is the most frequent site involved with diverticula and hence diverticulosis. Obstruction occurs in two scenarios.

One is due to the narrowing of bowel wall due to hypertrophy of the muscular layer. This type rarely causes complete obstruction but may be a cause of diagnostic challenge as these strictures are impossible to differentiate from neoplastic strictures. It may be difficult to pass a colonoscope through the stricture and hence resection may be the last resort to rule out a neoplastic lesion. This is also therapeutic.

The more common type is where the small bowel gets adhered to the phlegmon or an intra-abdominal abscess secondary to the infectious and inflammatory aspect of the diverticular disease. Treatment is by nasogastric decompression and antibiotics. An Intraabdominal abscess needs to be drained percutaneously.

Diverticular disease may also cause obstruction due to formation of enteroliths which cause luminal obstruction.

2. Crohn’s disease

Intestinal obstruction in Crohn's disease can be caused by

a. Active inflammation
b. Fibrotic stricture from chronic disease, or an
c. Abscess or phlegmon causing a mass effect with adhesion of bowel loops to this inflammatory process.
d. Chronic fibrosing lesions, which eventually cause narrowing of the bowel lumen, produces partial or near-complete obstruction.

Adhesions from previous abdominal operations are also a frequent cause of obstruction. Obstruction typically involves the small intestine, although large bowel obstruction from strictures may occur. Initial treatment includes bowel rest, nasogastric decompression, intravenous fluids, and anti-inflammatory medications, usually steroids. Obstruction caused by a stricture may be treated by endoscopic balloon dilation. A laparotomy may be needed if the above management fails. The treatment of choice of intestinal obstruction in patients with Crohn's disease

is segmental resection of the involved segment with primary re-anastomosis. This may involve segmental resection and primary anastomosis of a short segment of ileum if this is the site of the complication. More commonly, the cecum is involved contiguously with the terminal ileum, in which case resection of the involved terminal ileum and colon is required and the ileum is anastomosed to the ascending or transverse colon. Short segment stricture needs resection anastomosis while a long segment stricture is treated by stricturoplasty. This helps to preserve bowel length in cases of extensive disease.

3. Ulcerative Colitis

   This also can cause intestinal obstruction by predisposing to strictures and causing fistulae.

POSTOPERATIVE OBSTRUCTION [4, 9]

Early postoperative intestinal obstruction is defined as that which occurs within first 6 weeks postoperatively.

Early postoperative obstruction is mostly due to physiologic ileus but may also be secondary to mechanical causes. Such causes include presence of mesenteric defects, bowel having inadvertently been included in the abdominal wall closure. Early postoperative obstruction is most often managed conservatively though the potential risk of strangulation still exists even in this setting. Laparotomy is indicated when signs of complete obstruction persist beyond 3-4 days.

Second category of postoperative obstruction is that which develops 10 days to 4 weeks after a laparotomy. This is the most dangerous time for re-operation as adhesions may be extremely thick dense and highly vascular; and surgical intervention is frequently complicated by fistula formation. Non-operative therapy is the treatment modality of choice. Consideration must be given for a prolonged gastric decompression using percutaneous endoscopic gastrostomy or tube pharyngostomy with parenteral nutritional support. In some cases spontaneous relief occurs as the adhesions soften and mature.

INTUSSUSCEPTION IN ADULTS [4, 8, 9]

Telescoping of one segment (Intussusceptum) into another segment (Intussuscepiens) is called intussusception. This may result in ischemic injury to either of the segments. Colo-colic intussusception is more common in adults in contrast to the ileo-colic intussusception in children. Most have an underlying pathologic process called as the lead point. Initially bowel movements may be normal but, later on is characterized by evacuation of blood and mucus, classically described as the 'redcurrant jelly' stools [4]. A lump that hardens on palpation associated with emptiness in the right iliac fossa may be observed, called as the 'Sign of Dance' [4]. Except for the intestinal obstruction no other clinical features or radiologic imaging are specific enough for intussusception. X-rays may reveal absent gas shadows, with the characteristic claw sign on contrast enema.

Ultrasonography may reveal a concentric ring appearance called as the target sign or the bull's eye appearance [11]. CT scan also shows multiple concentric ring appearance.

Unlike in children, most adults with intussusception need celiotomy in view of the high frequency of intrinsic abnormalities of bowel such as polyps, submucosal lipoma or tumor causing the intussusception.

Hydrostatic reduction must not be attempted unlike in children where it is often used. At laparotomy, the intussusceptum must be gradually reduced, viability of the segment assessed. In case of non-viable segment a resection is done with primary anastomosis.

VOLVULUS [4, 9]

Axial rotation of a loop of bowel more than 180 degrees around the mesentery causing mechanical luminal obstruction is termed as volvulus. Most common site is the sigmoid colon. It has also been observed in caecum, stomach, large bowel and gall bladder. It is said to be primary when no predisposing anatomic factors are recognized. Secondary volvulus occurs around a well-defined point of fixation such a band or adhesion, Meckel's diverticulum or hernia, which fixes the mesentery but allows movement of proximal and distal bowel loops.

Volvulus by definition forms a closed loop obstruction with a high risk of strangulation. Symptoms are dramatic in onset with pain abdomen and abdominal distension. Radiologic appearance is of a bent inner tube in case of sigmoid volvulus commonly described as a coffee bean appearance, but other anatomic locations do not have any specific features on radiographs. They are best diagnosed only with a high index of clinical suspicion.

Initial treatment of sigmoid volvulus consists of endoscopic decompression where detorsion is indicated by a gush of faeces and air. Treatment is successful in 60% of cases but recurrence is common. Early recurrence is prevented by placing a rectal tube as a stent. Most patients need an elective resection of the redundant loop as a permanent solution. Those who fail endoscopic treatment are treated by a laparotomy. The loop is manually detorsed and redundant loop of mesentery is to be excised. Any nonviable loop has to be resected. Some form of intestinopexy needs to be done to prevent recurrence in those in whom resection is not performed. Any recognizable anatomic lead point such as bands or adhesions needs to be divided and corrected.

COMPOUND VOLVULUS [4]

This is a rare variety of volvulus where a long pelvic mesocolon allows the ileum to twist around the sigmoid colon, resulting in gangrene of either or both segments of bowel. The patient presents with acute intestinal obstruction. Treatment is with an emergency laparotomy, with decompression and if required, resection and anastomosis.

Fig 27 Sigmoid Volvulus

Fig 28 Sigmoid Volvulus

NEOPLASMS [4, 7, 9]

Primary intra-abdominal neoplasms are a frequent cause of obstruction of both the small & large bowels. Frequently indicated neoplasms are colorectal, gastric, small bowel and ovarian. Morbidity and mortality are very high in this group of obstruction secondary to the malignancy and hence treatment needs to be individualised to the patient considering the life-expectancy and risk-benefit ratio of the planned treatment protocol.

The various ways in which a neoplasm may cause obstruction are

1. Intraluminal growth
2. Strictures
3. Lead point causing intussusception
4. Large growth causing external compression
5. Infiltration to adjoining tissues causing adhesion and acute kinking of bowel lumen

Obstruction is more often seen with annular and tubular varieties of colonic carcinoma. A resection of the tumour must be done where ever a sufficient clearance is possible with primary anastomosis even in an unprepared bowel. It is risky to fashion an anastomosis in the presence of on-going intra-abdominal sepsis and highly distended bowel. If

emergency situations do not permit a primary anastomosis or even a resection, a Hartmann's procedure may be done and the proximal bowel must be brought out as a stoma to relieve the intestinal obstruction and a second surgery may be planned at a later date when the patient becomes more stable physiologically to tolerate an extensive surgery.

Fig 29 Sub-acute obstruction due to growth in ascending colon

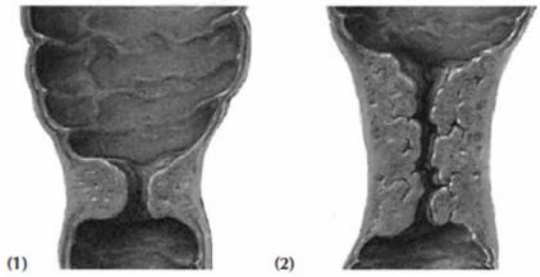


Fig 30 Common types of intestinal neoplasms causing bowel obstruction 1, Annular, 2-Tubular

STRICTURES [4, 9]

Strictures are an intramural cause of mechanical intestinal obstruction. These strictures may be secondary to

1. Congenital
2. Inflammatory diseases such as Crohn's disease
3. Infective lesions as tuberculosis and typhoid
4. Neoplastic strictures
5. Post anastomotic strictures

Strictures are difficult to diagnose on clinical features or investigations alone. They may be diagnosed at laparotomy or diagnostic laparoscopy. Short segment strictures whether single or multiple strictures at short intervals are usually treated by resection of the structuring segment. However long segment strictures and multiple strictures spaced far apart as in Crohn's disease, are treated by stricturoplasty of the involved intestines.

Fig 31 Stricture in the Jejunum

TUBERCULOSIS ABDOMEN [4, 9]

It can affect any part of the gastrointestinal tract most commonly involving the ileum and proximal colon. Various forms of intestinal tuberculosis have been described such as –

1. Ulcerative,
2. Hypertrophic
3. Ulcerohypertrophic

Intestinal obstruction is more often seen in the hyperplastic type. Presentation may be with an inflammatory mass an abdomen, stricturing disease and fistula formation. Main clinical features are with pain abdomen and distension secondary to intestinal obstruction. Treatment is mostly medical in cases without intestinal obstruction with resection of the obstructed segment needed in case of strictures and fistulae.

Other infectious causes of intestinal obstruction are typhoid strictures which frequently occur in the convalescent period at the sites of typhoid ulcers.

INTRALUMINAL CAUSES OF OBSTRUCTION [4, 7, 9]

Intraluminal obstruction can be caused by various entities such as

1. Gall stones

   This occurs in the elderly, secondary to the erosion of a gall stone through the gall bladder to the duodenum. This commonly gets impacted in the ileum about 60cm proximal to the ileo-caecal valve. Patient has recurrent attacks of subacute obstruction due to the ball valve effect of the stone. Diagnostic finding in the radiograph is the air fluid level in the biliary tree and the stone per se may not be visualized. Treatment is by crushing the stone at laparotomy after milking it proximally. Sometimes, the bowel may have to be opened to remove the stone. The region of gall bladder must not be explored.

2. Food bolus

   This frequently occurs after a partial gastrectomy when food items pass into the bowel before being down into smaller particles. Treatment is similar to that of gall stone obstruction by either crushing and milking or enterotomy and removal.

3. Phytobezoar

   Phytobezoars may result from a high fibre intake, inadequate chewing, previous gastric surgery, hypochlorhydria and loss of

the gastric pump mechanism. When possible, the lesion may be kneaded into the caecum, otherwise open removal is required.

4. Trichobezoar

   These are firm masses of undigested hair balls and fruit/vegetable fibre respectively. The former is due to persistent hair chewing or sucking, and may be associated with an underlying psychiatric abnormality.

5. Strecolith

   These are usually found in the small bowel in association with a jejunal diverticulum or ileal stricture. Presentation and management are identical to that of gallstones.

6. Worms

   This is commonly seen in children, institutionalized people and in tropic regions. An attack frequently follows initiation of anti-helminthic therapy. Diagnosis is by either sighting worms in the stools or on a radiograph within the dilated small bowel loops and where this is not possible, diagnosis may be suggested by eosinophilia. Treatment is by laparotomy and kneading the tangled mass into the caecum; if not it should be removed. Occasionally, worms may cause a perforation and peritonitis, especially if the enteric wall is weakened by such conditions as ameobiasis.

PSEUDO-OBSTRUCTION (Ogilvie's Syndrome) [4, 7, 9]

Though pseudo-obstruction is not a cause of mechanical obstruction, this entity often causes diagnostic challenge in intestinal obstruction. The exact pathogenetic mechanism has not yet been clearly described. Various factors associated with pseudo-obstruction are

Idiopathic

1. Metabolic
    a. Diabetes: intermittent porphyria
    b. Acute hypokalaemia
    c. Uraemia
    d. Myxoedema
2. Severe trauma (especially to the lumbar spine and pelvis)
3. Shock as in Burns, Myocardial infarction and Stroke

Septicaemia

1. Retroperitoneal irritation by Blood, Urine, Enzymes (pancreatitis) and Tumour
2. Drugs as Tricyclic antidepressants, Phenothiazines, Laxatives

3. Secondary gastrointestinal involvement as in Scleroderma and Chagas' disease

Primary pseudo-obstruction is a motility disorder which is either a familial visceral myopathy (hollow visceral myopathy syndrome) or a diffuse motility disorder involving the autonomic innervation of the intestinal wall. Secondary pseudo-obstruction is more common and has been associated with Parkinson's disease, neuroleptic medications, myxoedema, diabetes mellitus, uraemia, hyperparathyroidism, lupus, opiates, severe metabolic illness, scleroderma, and traumatic retroperitoneal hematomas.

Sympathetic over activity overriding the parasympathetic system has been considered to play an important role. Treatment with neostigmine, a parasympathetic drug also supports this theory. Further supporting evidence is by immediate resolution of the syndrome after administration of an epidural anaesthetic that provides sympathetic blockade. Patient often presents with abdominal distension. Abdomen is usually tympanic, non-tender and bowel sounds are heard. The most useful diagnostic investigation is a water soluble contrast enema which differentiates this entity from mechanical obstruction. Treatment is by conservative management, and neostigmine which causes resolution soon after drug administration.

METHODS AND MATERIALS

An observational study was conducted in Coimbatore Medical College Hospital from September 2011 to November 2012 among adult Patients being admitted to the surgical wards of Coimbatore Medical College Hospital with symptoms and/or signs of Intestinal obstruction.

Inclusion Criteria:

1. Patients presenting to the hospital both to the regular OPD and to the emergency department with symptoms and/or signs of Intestinal obstruction subsequently undergoing admission
2. Age of patients > 12 years

Exclusion Criteria:

1. Paediatric patients
2. Patients who were not willing for admission to the hospital
3. Patients diagnosed with intestinal obstruction in the immediate postoperative period as in Paralytic ileus
4. Patients having a non-mechanical cause of intestinal obstruction

The patients presenting to the hospital were initially resuscitated hemodynamically. Detailed history with special importance to the

previous history of similar complaints; history of previous abdominal surgeries; co-morbid factors were recorded. Associated metabolic derangements were corrected to near normal levels. The Patients were investigated with routine blood investigations and also radiologically with X-ray and/or CT scan study of the abdomen. However in acute emergencies only the basic investigations for diagnosis were employed initially with patients undergoing full evaluation subsequently where necessary. Patients were followed up till the time of discharge to look for any early post-operative complications. Details of the treatment along with Intra-operative findings as well as post-operative complications were recorded. Analysis of the various causes of mechanical bowel obstruction and the outcome following management of the patients was done using the collected data.

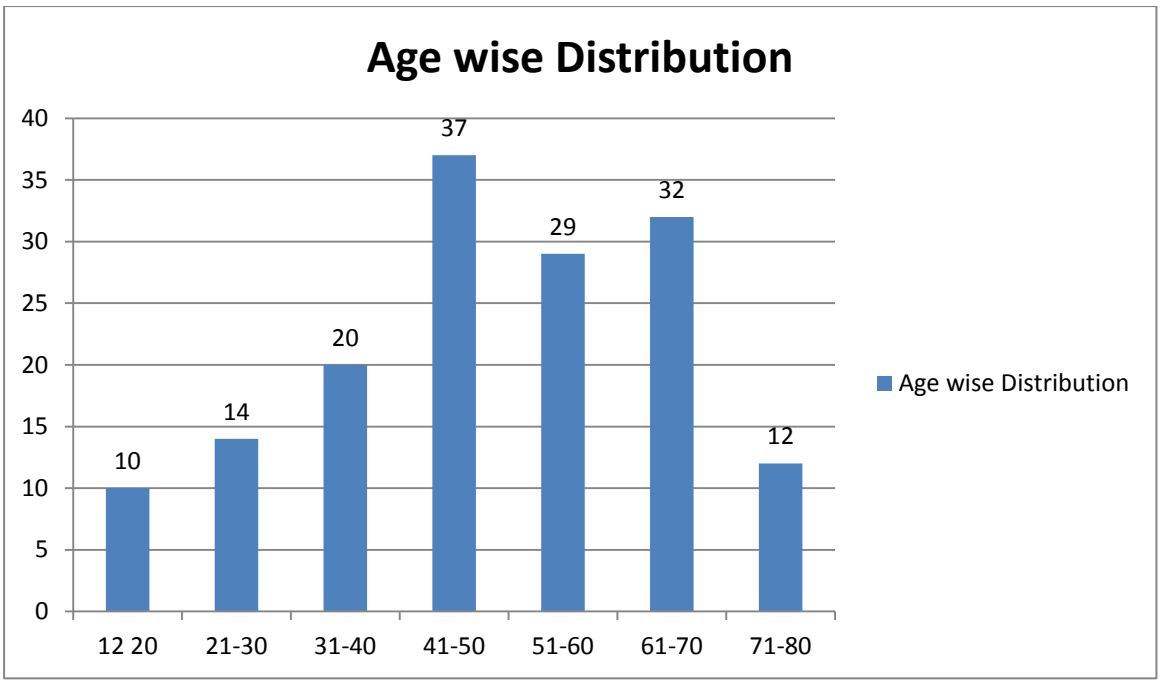
OBSERVATIONS & DISCUSSION

In our study on mechanical intestinal obstruction conducted from September 2011 to November 2012 at Coimbatore Medical College Hospital, 154 adult cases of Mechanical intestinal obstruction undergoing surgical intervention were included in the study. During the study, patients were resuscitated adequately and taken up for surgery. Patient details were collected using a standard proforma. The details were then compiled into a master chart and following observations made.

1. AGE DISTRIBUTION OF PATIENTS

Age Group (years)	No of patients	Percentage
12-20	10	6.49
21-30	14	9.09
31-40	20	12.98
41-50	37	24.02
51-60	29	18.83
61-70	32	20.77
71-80	12	7.79

Majority of the patients in the study were found to be middle aged adults aged within 40 to 50 years. Mean age of presentation was 48.88 years.



AGE DISTRIBUTION OF PATIENTS

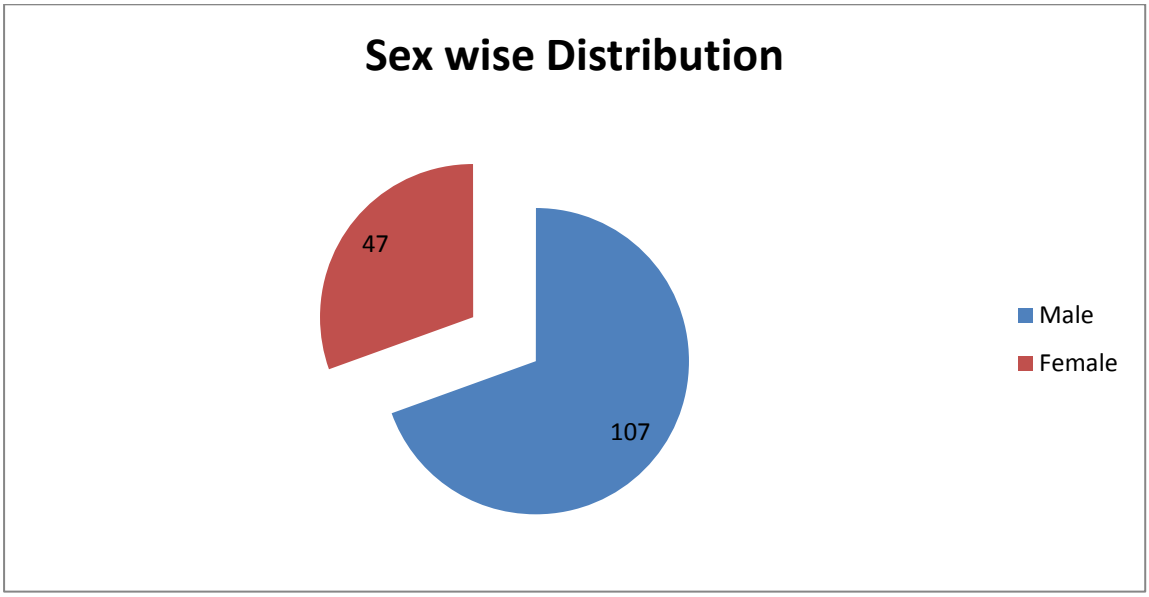
The mean age distribution of our study population corresponds to other studies as

1. *'The mean age of the study population was 43.08 ± 13.07 years.'* - ***Pattern of Acute Intestinal Obstruction: Is There a Change in the Underlying Etiology?,*** *Arshad M. Malik, Madiha Shah, Rafique Pathan, and Krishnan Sufi, Saudi J Gastroenterol. 2010 October; 16(4): 272–274*

2. 'The commonest age group affected was 20-60 years.' - ***'Etiology and Outcome of Acute Intestinal Obstruction: A Review of 367 Patients in Eastern India';*** *Adhikari Souvik, Mohammed Zahid Hossein, Das Amitabha, Mitra Nilanjan,1 and Ray Udipta; Saudi J Gastroenterol. 2010 October; 16(4): 285–287.*

2. SEX WISE DISTRIBUTION OF PATIENTS

Sex	No. of cases	Percentage
Male	107	69.48
Female	47	30.52



Majority of the patients were male adults (106) as opposed to 48 females. This huge gender discrepancy is probably due to the proportion of hernia which is mainly seen in males and is the most common etiology in the study.

SEX WISE DISTRIBUTION OF PATIENTS

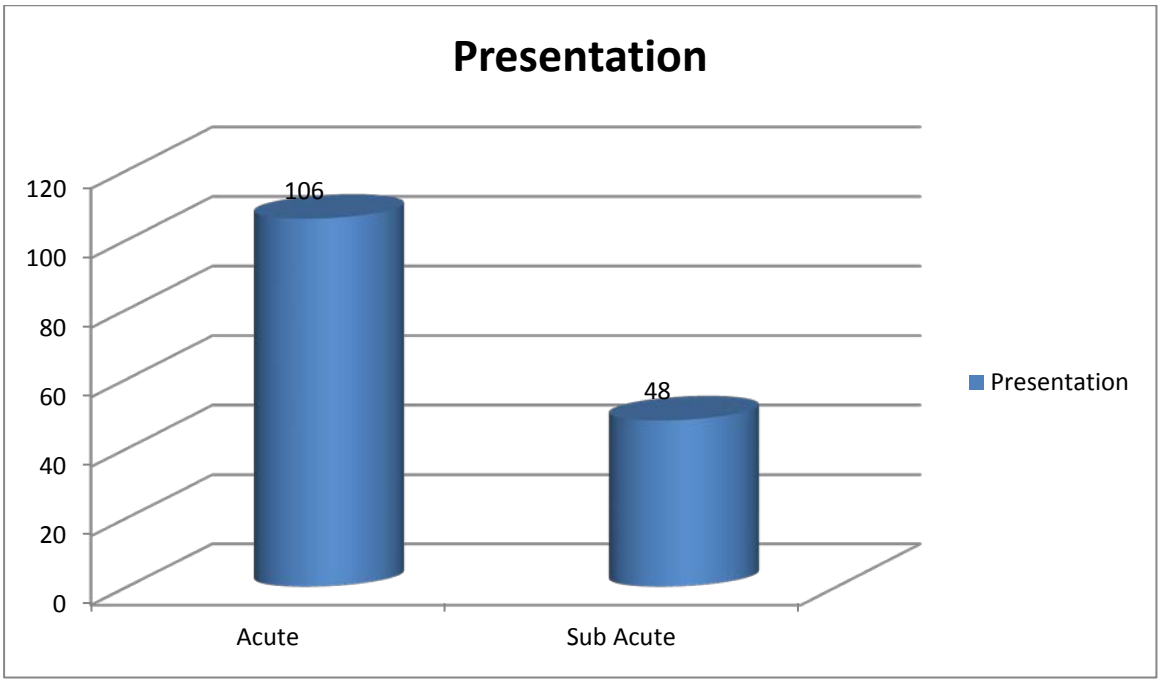
Other journals also support this evidence of mechanical intestinal obstruction being more common in males.

1. *'A total of 100 patients were treated for mechanical bowel obstructions during the study period. There were 83 males and 17 female. Male to female ratio was 5:'* ***- Analysis of different causes of mechanical intestinal Obstruction,*** *Issue Year : 2009, Issue Number : 5, Issue Month : December, Muhammad Saeed Akhtar, Irfan Shukr*

2. *The gender discrepancy in our patients with males outnumbering females by a huge margin can be possibly accounted for, as a large number of our patients had obstructed inguinal hernia, and in our country we mostly have males who suffer from this condition.* ***'Etiology and Outcome of Acute Intestinal Obstruction: A Review of 367 Patients in Eastern India';*** *Adhikari Souvik, Mohammed Zahid Hossein, Das Amitabha, Mitra Nilanjan,1 and Ray Udipta; Saudi J Gastroenterol. 2010 October; 16(4): 285–287.*

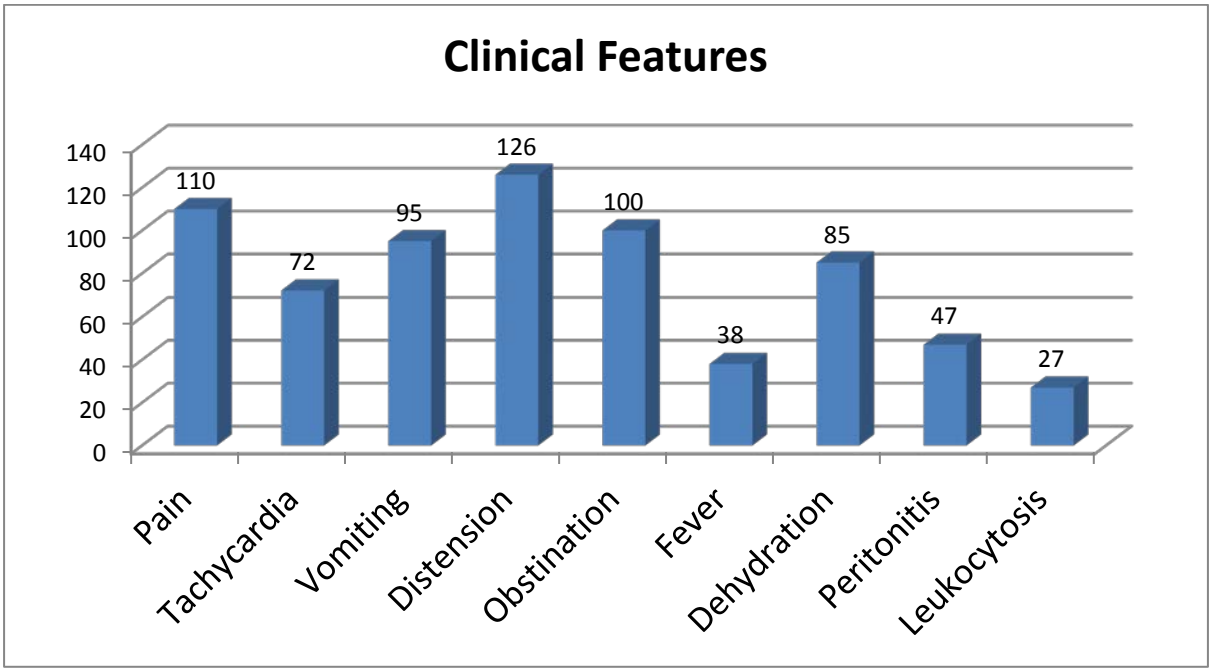
3. MODE OF PRESENTATION

Most of patients encountered were acute in presentation in a ratio nearing 2:1as compared to subacute presentation. Most patients in our study presented as acute intestinal obstruction requiring emergency surgical intervention.



4. CLINICAL FEATURES

The cardinal features of intestinal obstruction were altered bowel habits, Pain, Distension of abdomen and Vomiting. The most predominant clinical feature among patients in our study was abdominal distension commonly referred as to postprandial distension. Pain was the next in frequency and was more in those cases of acute presentation than subacute presentation. Among patients presenting acutely, pain and abdominal distension were the most frequent complaints. More than 40% patients had vomiting. It was bilious in nature in 46 patients and 18 patients had a feculent vomiting. It was observed that more the number of the above mentioned symptoms/signs in a patient, more were the risk of strangulating obstruction.



CLINICAL FEATURES

Other studies have also shown similar incidence of presenting complaints as in

1. *The most common features on presentation included distension of the abdomen (87%), pain in abdomen (78%), vomiting (73%), absolute constipation (88%), and dehydration (67%).* ***Pattern of Acute Intestinal Obstruction: Is There a Change in the Underlying Etiology?,*** *Arshad M. Malik, Madiha Shah, Rafique Pathan, and Krishan Sufi, Saudi J Gastroenterol. 2010 October; 16(4): 272–274*

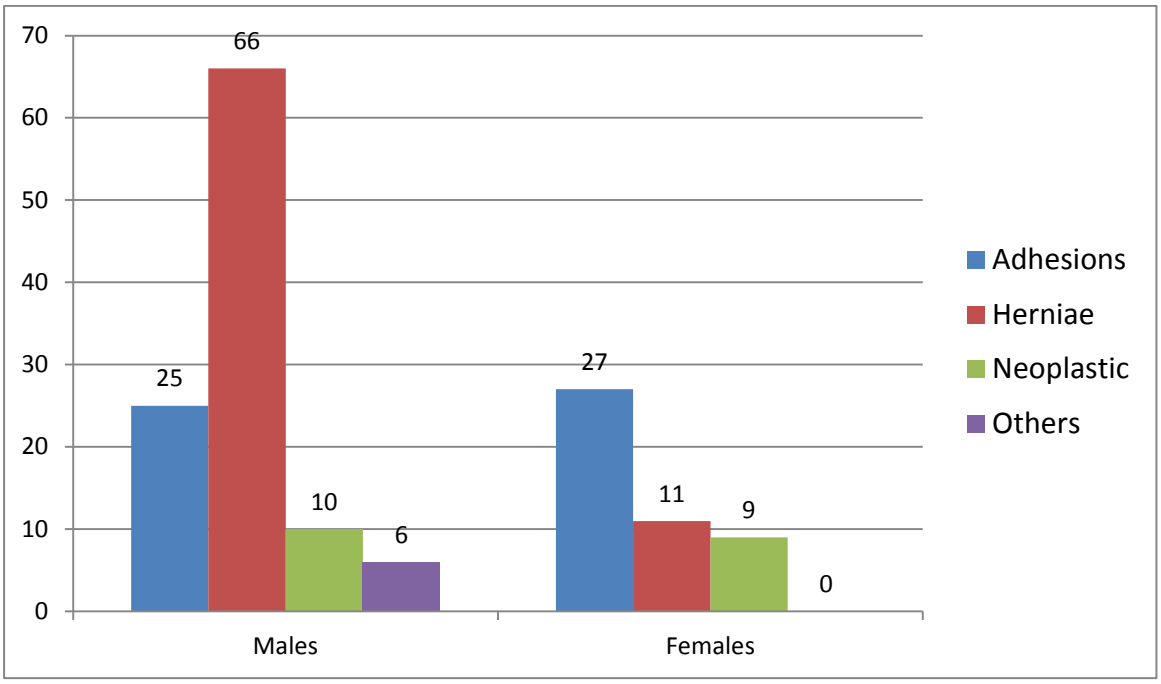
2. *Criteria for admission were obstipation (279 patients: 76.02%), colicky pain (263 patients: 71.66%), abdominal distension (341 patients: 92.92%), and nausea and vomiting (91 patients: 24.8%).'* - ***'Etiology and Outcome of Acute Intestinal Obstruction: A Review of 367 Patients in Eastern India';*** *Adhikari Souvik, Mohammed Zahid Hossein, Das Amitabha, Mitra Nilanjan,1 and Ray Udipta; Saudi J Gastroenterol. 2010 October; 16(4): 285–287.*

5. LAPAROTOMY FINDINGS IN STUDY POPULATION

In our study, the most common cause of mechanical intestinal obstruction was external hernias, followed by intra-abdominal adhesions. Extraluminal causes were detected in 128 patients, mural causes in 7 and intraluminal causes in 20 patients. Among male patients, hernia was the most common cause of obstruction whereas adhesions formed the largest cause among the female patients. During the study, we encountered 2 cases of Sigmoid volvulus where a resection – anastomosis was done. One case of sigmoid volvulus succumbed to pre-operative long standing metabolic alterations.

	Male	Percentage	Female	Percentage
Adhesions	25	23.36	27	57.44
Hernia	66	61.68	11	23.40
Neoplastic	10	9.34	9	19.14
Others	6	5.61	0	0.0

LAPAROTOMY FINDINGS IN STUDY POPULATION



This is in contrast to data from most western series probably due to

1. Delay in presentation and treatment of the common hernias
2. Higher frequency of abdominal surgeries in developed nations
3. Better awareness leading to early elective treatment of herniae before it can complicate as obstruction

LAPAROTOMY FINDINGS IN STUDY POPULATION

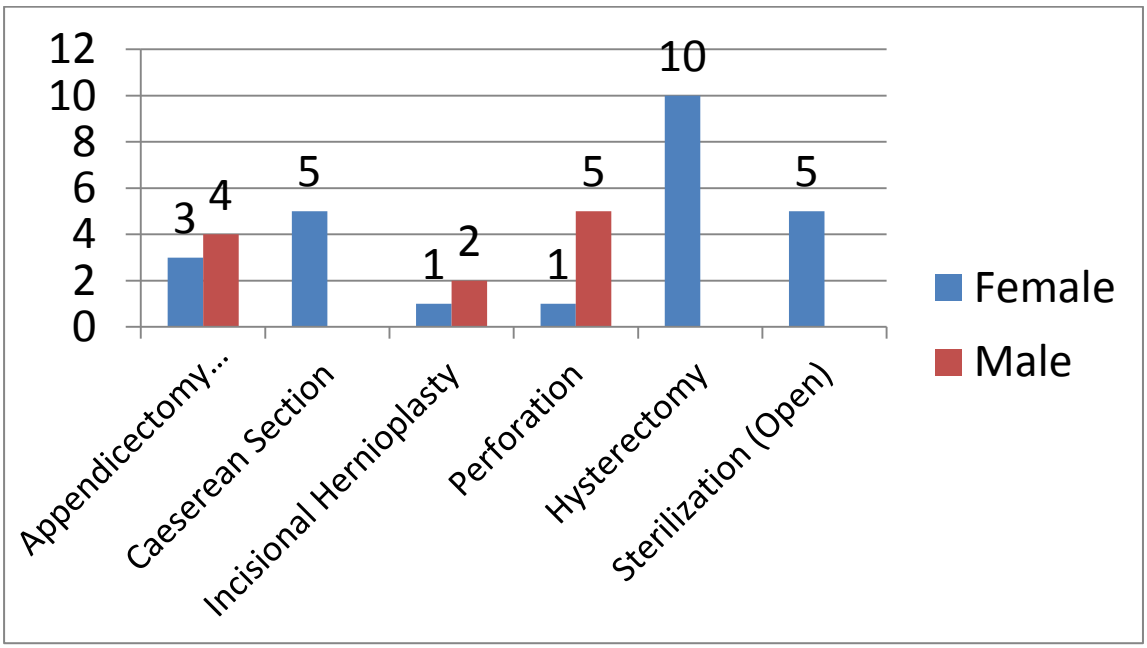
However our data is consistent with many papers published on the similar topic in the less developed parts of the world.

1. *'This study demonstrates that the pattern of intestinal obstruction differs from the Western world with obstructed hernias being the most important cause and also emphasizes the fact that intestinal tuberculosis assumes a prominent role.'-* ***'Etiology and Outcome of Acute Intestinal Obstruction: A Review of 367 Patients in Eastern India';*** *Adhikari Souvik, Mohammed Zahid Hossein, Das Amitabha, Mitra Nilanjan and Ray Udipta; Saudi J Gastroenterol. 2010 October; 16(4): 285–287.*

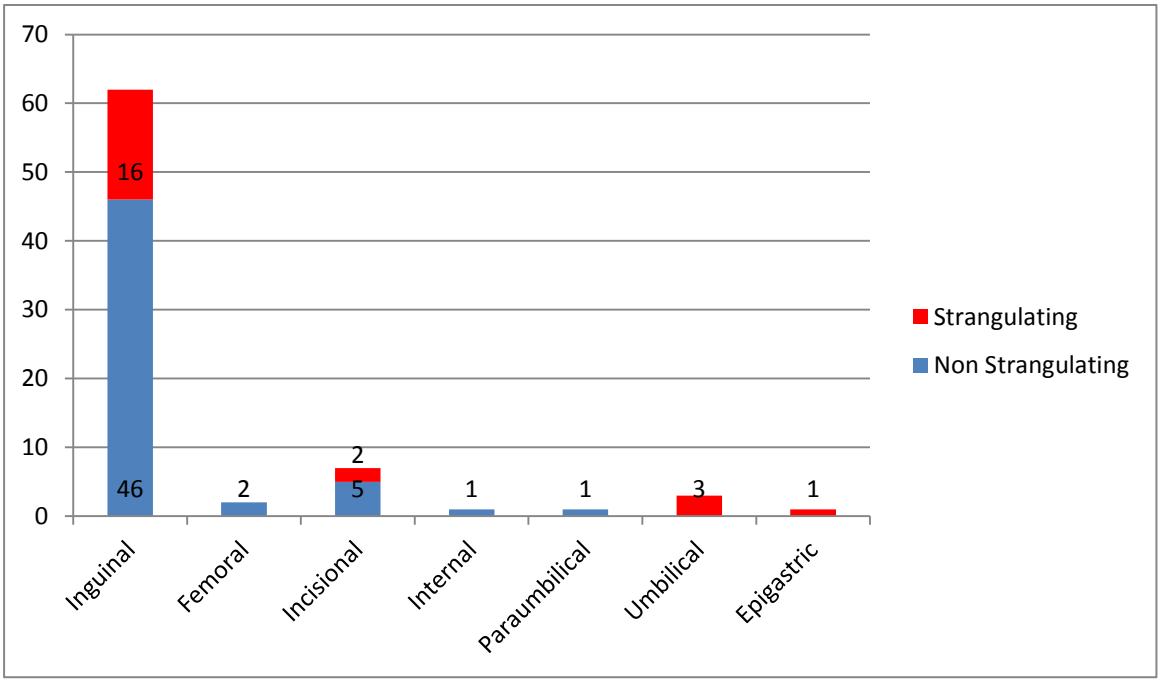
2. *'Despite many improvements in the health care setup, obstructed/strangulated hernias constitute commonest cause of intestinal obstruction in our setup, depicting a delay in diagnosis and treatment of this common surgical problem. Countries with the well-developed health care system, Adhesions are the commonest cause of intestinal obstruction'. –* ***'Pattern of intestinal obstruction a hospital based study'****; Jahangir Sarwar Khan, Junaid Alam, Hamid Hassan, Mohammad Iqbal; Pak AF Med J. 2007 December, 4(1)*

6. ADHESIVE INTESTINAL OBSTRUCTION

Among the 52 adhesive intestinal obstructions, we found that 16 were spontaneous adhesions and remaining 36 were post-operative adhesions. The most frequently implicated index surgery was Hysterectomy (27.77%) followed by open appendectomy (19.44%), Caesarean section (13.88), perforation surgery (16.67), and open sterilization procedures (13.88%). These figures are in conjunction with various other studies which have implicated surgeries in the infra-mesocolic compartment to be more frequently associated with adhesive intestinal obstruction. There were also cases of adhesions due to in inflammatory pathology in the peritoneum such as appendicitis (6), abdominal tuberculosis (6), pancreatitis (2), peritonitis (1) and one was spontaneous where no primary cause could be identified.



7. HERNIAS IN INTESTINAL OBSTRUCTION

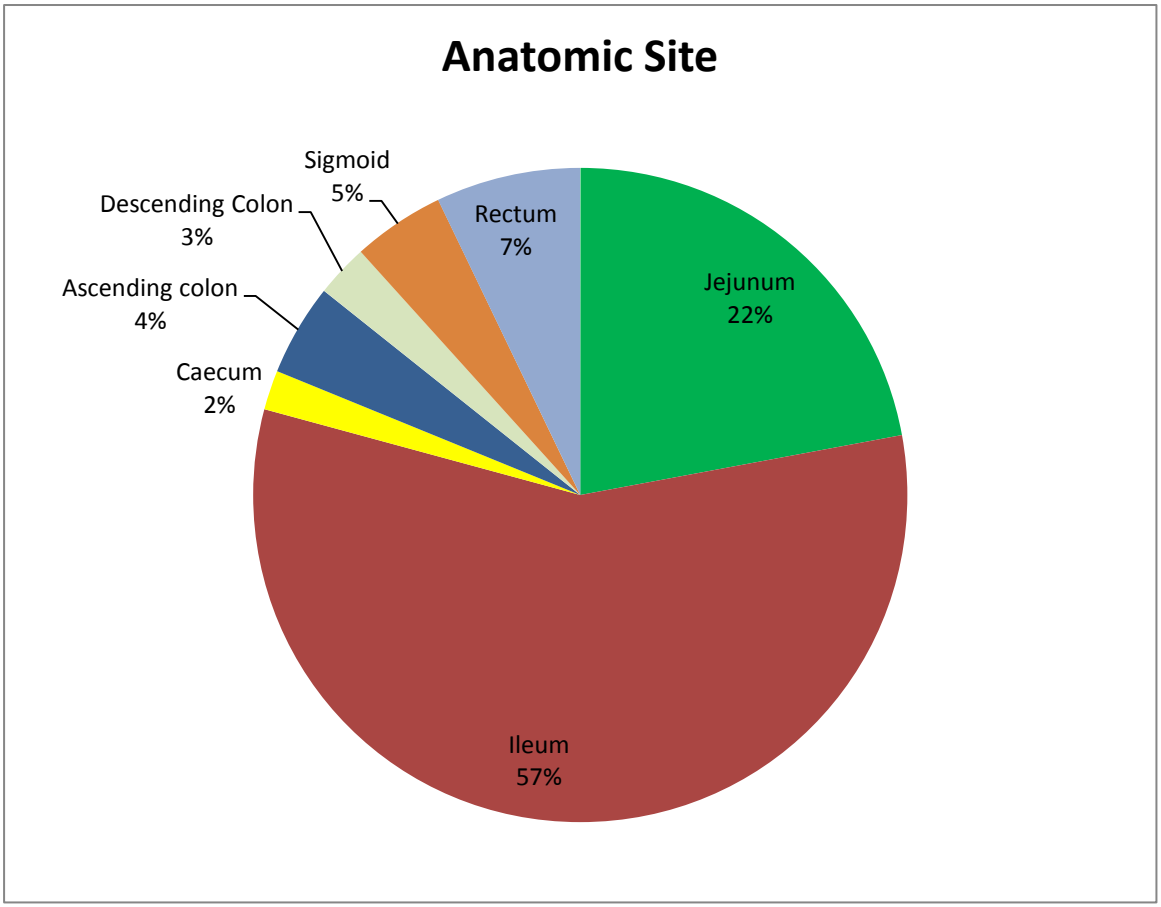


Among all the hernias causing intestinal obstruction, inguinal hernia was the most common (80.51%), followed by incisional (9.09%), umbilical (3.89%) femoral hernias (2.5%), epigastric (1.2%), para-umbilical (1.2%) and internal hernia (1.2%).

Of the hernias, we had 16 strangulated inguinal hernias, 3 strangulated umbilical hernias, 2 strangulated incisional hernia and 1 strangulated epigastric hernia. In these cases, due to the non-viability of the bowel, a resection and primary end to end anastomosis was done. Hernioplasty in such cases carried a risk of mesh infection.

8. ANATOMICAL SITE OF OBSTRUCTION

Small bowel superseded the large intestines in frequency of getting obstructed. The relative frequency of various anatomic sites of obstruction were Ileum (57%), Jejunum (22%), Rectum & Sigmoid (12%), Ascending colon (4%), descending colon (3%) and caecum (2%). A vast majority of obstructions were in the ileum of all causes put together. Most of these were adhesive in nature followed by trapping of the bowel in a hernial sac. Large bowel obstruction was mainly secondary to neoplastic causes while in small bowel it was due to hernia and adhesions.



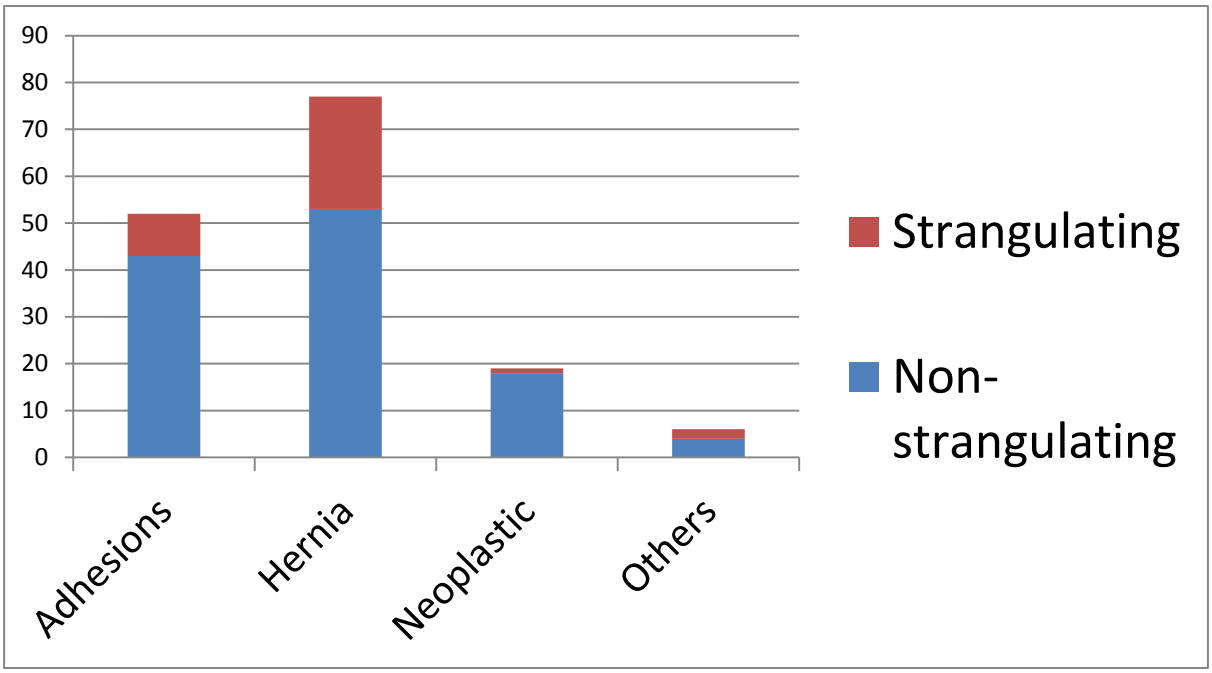
ANATOMICAL SITE OF OBSTRUCTION

Other similar studies have also implicated small bowel as being more commonly involved in mechanical obstruction.

1. *'Site of obstruction was found to be in the small bowel in a vast majority (85%) of the study population; while in 35 (15%) patients, the site of obstruction was in the large bowel.'* - ***Pattern of Acute Intestinal Obstruction: Is There a Change in the Underlying Etiology?,*** *Arshad M. Malik, Madiha Shah, Rafique Pathan, and Krishan Sufi, Saudi J Gastroenterol. 2010 October; 16(4): 272–274*

9. STRANGULATING vs. NON-STRANGULATING OBSTRUCTION

In the study, most of the cases observed were non strangulating obstruction. Among the strangulating obstruction, hernia was the most frequent precipitating factor contributing to more than 70.58% of the strangulating obstruction. Among the 52 adhesive intestinal obstructions, 9 of them were strangulated obstruction. Inguinal hernia topped the list of strangulating hernia as inguinal hernia was more common compared to the other types of hernia in our study.



10. CLINICAL FEATURES IN PREDICTING RISK OF STRANGULATION

Various studies have suggested the importance of preoperative evaluation in the diagnosis & differentiation of Strangulating from non strangulating intestinal obstruction. In our study, we evaluated the importance of Persistent pain, fever, tachycardia, features of peritonitis and leukocytosis in predicting the risk of strangulating obstruction. It was found that when more than 3 of the above factors were present, it had a positive predictive value of about 78.33% and when more than 4 of the above factors were present, the positive predictive value approached 93.54%. However none of the factors are sufficiently specific or sensitive to predict the risk of strangulation preoperatively. This reinforces the importance of repeated clinical evaluation of the patient and having a high index of clinical suspicion to accurately predict the occurrence of strangulating obstruction and timely surgical intervention where necessary.

Number of C/f	Strangulating	Non strangulating	Positive predictive value
>3	32	9	78.04%
>4	28	2	93.33%
>5	19	1	95.00%

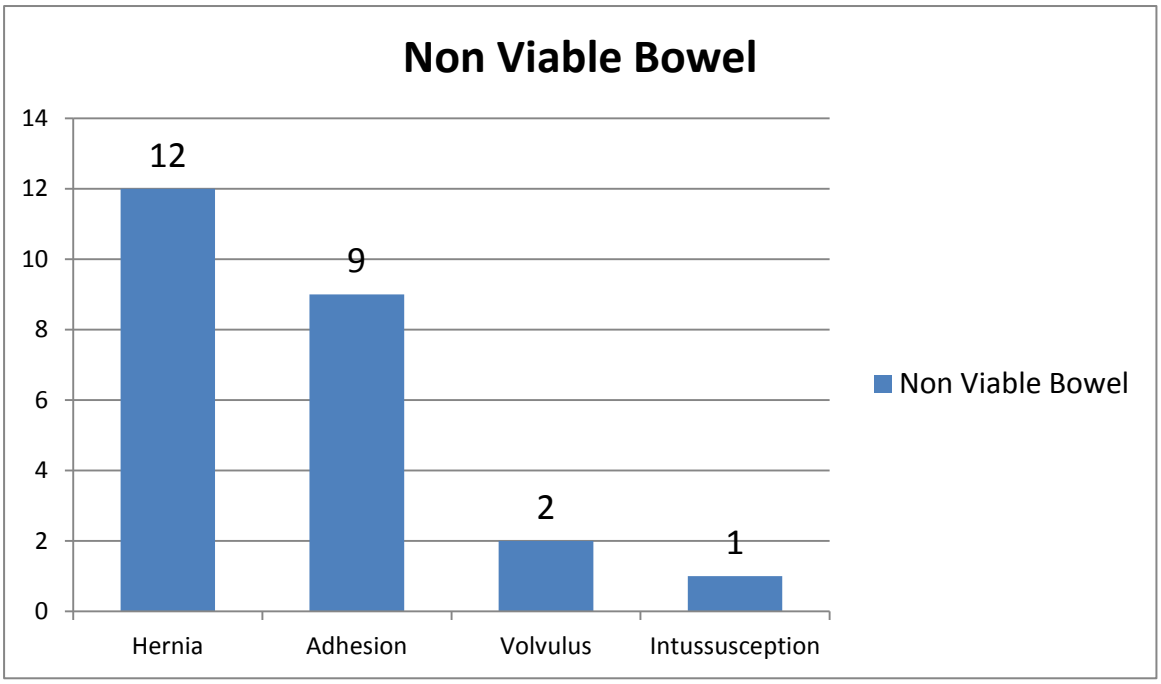
Other studies that have evaluated pre-operative prediction of the risk of strangulation suggest that:

1. ***'Overall, the preoperative assessment was correct in only 35 of the 51 patients (efficiency, 70 percent). These data show that in patients with complete mechanical small bowel obstruction, the preoperative diagnosis of strangulation cannot be made or excluded reliably by any known clinical parameter, combination of parameters, or by experienced clinical judgement.',*** *Sarr MG, Bulkley GB, Zuidema GD, Preoperative recognition of intestinal strangulation obstruction. Prospective evaluation of diagnostic capability, Am J Surg 1983 Jan; 145(1):176-82.*

11.BOWEL VIABILITY

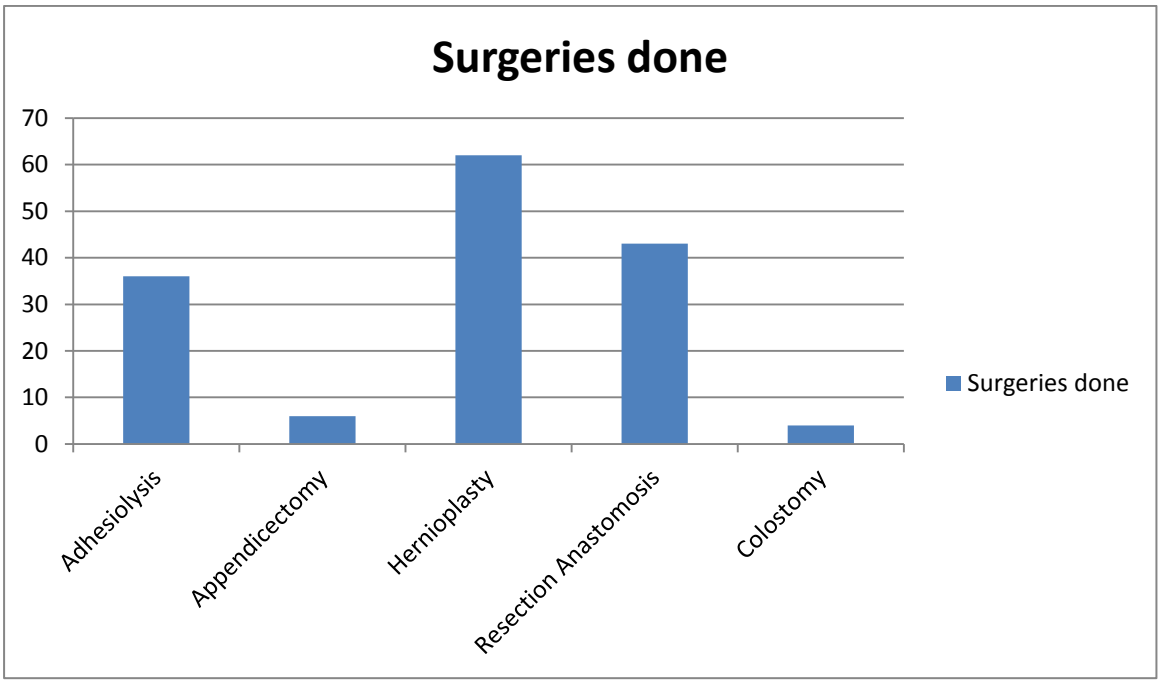
The bowel was found to be non-viable in 24 cases. About half the cases of where bowel was found non-viable were due to external hernia (12), other causes being adhesions, volvulus and one intussusception. All these cases needed resection of the nonviable bowel with end to end anastomosis. Among these patients undergoing resection-anastomosis, the complications seen were

1. Anastomotic leak - 2
2. Post-operative wound infection – 4
3. Death - 5



12. TREATMENT MODALITY USED

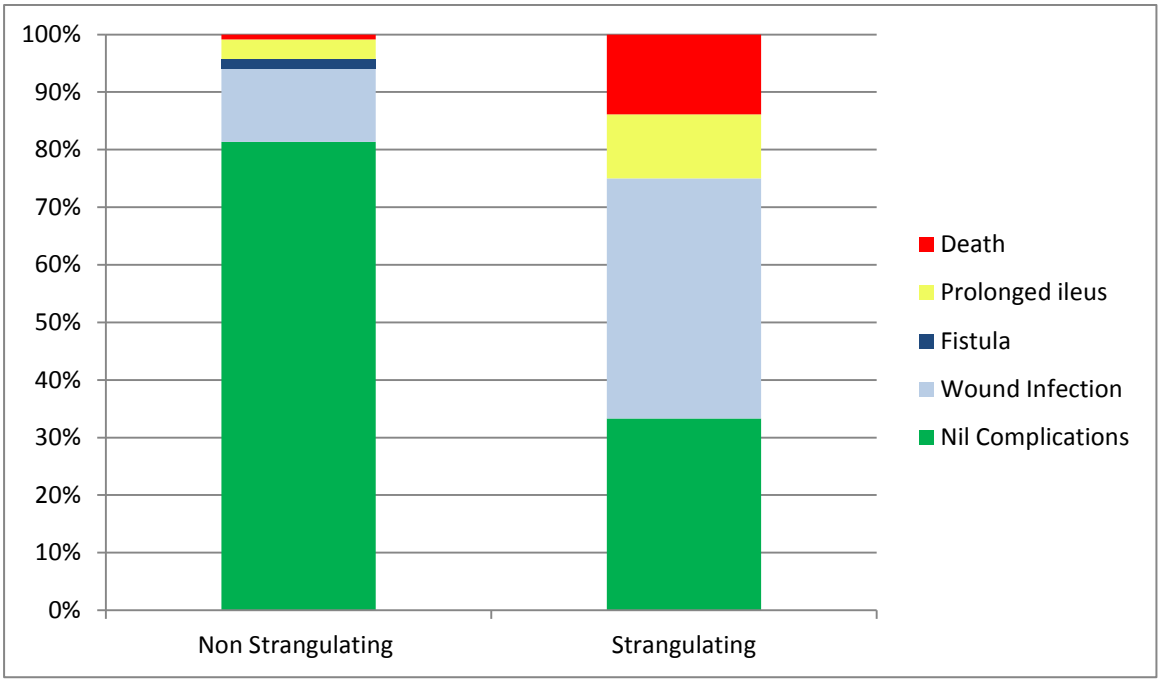
Hernioplasty was the most frequently performed procedure in our study. Wherever resection anastomosis was done in strangulated hernia, repair of hernia defect was done with a calculated risk of infection. 20 of the 43 resection anastomosis were done for non-strangulating diseases such as neoplastic lesions and intra mural pathologies as strictures. In neoplastic cases presenting acutely where a resection anastomosis was not deemed feasible, a colostomy was done. Appendectomy along with adhesiolysis was done in 7 patients where intraabdominal adhesions had developed secondary to acute appendicitis.



13. OUTCOME OF STRANGULATING vs. NON-STRANGULATING OBSTRUCTION

Strangulating obstruction was associated with a higher need for resection – anastomosis and a higher incidence of pre-operative metabolic alterations in the presentation. Hence risk of post-operative complications such as prolonged ileus, wound infection and mortality was more with strangulating obstruction. Overall morbidity and complications were more frequent in strangulating obstruction (66.67%) as opposed to non-strangulating obstruction (18.64). Average post-operative hospital stay secondary to complication was also more in the strangulating obstruction. Most frequently encountered complication was wound infection which occurred in about 41.67% of strangulating obstruction and in only 12.71% in non-strangulating obstruction. Mortality was also more in strangulating obstruction (13.89%) compared to non-strangulating obstruction (0.84%). These data are comparable to other similar studies which suggest 5-30% mortality in strangulating obstruction and also have demonstrated an increased morbidity in strangulating obstruction.

Outcome of strangulating vs. Non-strangulating obstruction



Post-operative complications	Non-Strangulating obstruction	Percentage (%)	Strangulating obstruction	Percentage (%)
Wound infection	15	12.71	15	41.67
Fistula formation	2	1.69	0	
Prolonged ileus	4	3.39	4	11.11
Death	1	0.84	5	13.89

SUMMARY

1. Mean age of the study population was 48.88 years with more males than females in the ratio of 2:1.

2. Acute presentation was more common than subacute types

3. Most common symptoms in the study group were distension of abdomen and pain abdomen

4. External hernias were the most common pathology followed by adhesions in our study population at Coimbatore Medical College Hospital in causing mechanical intestinal obstruction.

5. Among adhesive obstruction, lower abdominal surgeries & those on the infra-mesocolic compartment were more frequently implicated index surgeries. Meticulous care during surgery with regard to dissection techniques and gentle tissue handling helps to reduce post-operative adhesions.

6. Small bowel obstruction was more common than large bowel obstruction and ileum was the most often segment involved

7. Persistent pain, fever, tachycardia, peritonitis & leukocytosis which were having a positive predictive value of more than 95% when 4 or more factors were present, in predicting the risk of strangulating obstruction.

8. Strangulating obstruction is associated with a higher morbidity & mortality and hence the need for early recognition & prompt treatment

9. In our study the most common diagnostic modality used was plain X-ray abdomen in the acute sitting. The other investigatory modalities were ultrasonography, CT abdomen which further aided in arriving at an accurate pre-operative diagnosis. Wider application of these can help in better and early diagnosis of strangulating obstruction.

10. Initial conservative management may be tried in selected patients with careful observation of subtle signs of deterioration when conservative management is to be terminated.

11. Urgent surgical relief of obstruction is necessary to prevent irreversible damage to the intestines and homeostatic mechanism of the body. This also helps in reduction of the morbidity and mortality of patients.

12. Certain drawbacks are inherent in our study. Being a tertiary care hospital, the type of cases referred and handled in the institution are different and those which could not be managed at other levels of health care. Also most patients treated at our setup are from lower socio-economic group. Hence the etiologies and outcome seen in the study may not reflect the etiology in the general population.

CONCLUSION

The most frequent cause of intestinal obstruction is external herniation followed by adhesions.

Most frequent presentation is abdominal distension and pain.

Surgical intervention must be done at an appropriate time before complications arise

Morbidity, mortality and post-operative hospital stay are more in strangulating obstruction in comparison to the non-strangulating obstruction.

.

Case No.

Appendix-I

PROFORMA

Name: Age: Sex: IP No.:

Address:

D.O.Admission:

D.O.Surgery:

D.O.Discharge:

Duration of Illness:

History:

1. Pain Abdomen
2. Vomiting
3. Bowel Habits
4. Abdominal Distension
5. Fever
6. Loss of weight
7. Previous Surgery
8. Tuberculosis
9. Co-morbidities

GENERAL EXAMINATION
Pulse
Blood Pressure
Temperature
Hydration
Nutritional status

Abdominal Examination

1. Distension
2. Visible peristalsis
3. Scar
4. Tenderness
5. Rebound /Percussion Tenderness
6. Guarding
7. Rigidity
8. Bowel Sounds
9. Palpable Mass
10. Hernial Orifices
11. Digital Rectal Examination

Investigations

Hematological

1. Hb%
2. Total Count
3. Differential Count
4. B.Urea
5. B.sugar
6. S.Creatinine
7. S.Sodium
8. S.Potassium

Radiology

1. Chest X-Ray
2. X-ray Abdomen
3. USG Abdomen
4. CT Scan Abdomen

Diagnosis:

Operative Details

1. Findings

2. Procedure Done

Post-Operative Period

Biopsy Report

Follow Up

Appendix-II

BIBLIOGRAPHY:

1. Gray's Anatomy; The anatomical basis of Clinical practice, 39th Edition
2. Skandalaki's clinical anatomy, 5th edition
3. Ganong's Review of Medical physiology, 23rd ed.
4. Bailey and Love; Short Practice of Surgery, 25th ed; p no. 1188-04
5. Sabiston Textbook of Surgery; 18th ed
6. Josef E Fisher ; Mastery of Surgery;; 5th ed
7. Maingot;Abdominal operations; 11th ed
8. Hamilton Bailey; Emergency Surgery; 13th ed
9. Shackelford's surgery of the Alimentary tract; 5th ed
10. Grainger & Allison's Diagnostic Radiology, 4th ed.
11. Adhikari Souvik, Mohammed Zahid Hossein, Das Amitabha, Mitra Nilanjan,1 and Ray Udipta; Etiology and Outcome of Acute Intestinal Obstruction: A Review of 367 Patients in Eastern India; Saudi J Gastroenterol. 2010 October; 16(4): 285–287
12. Arshad M. Malik, Madiha Shah, Rafique Pathan, and Krishan Sufi;Pattern of Acute Intestinal Obstruction: Is There a Change in the Underlying Etiology?; Saudi J Gastroenterol. 2010 October; 16(4): 272–274
13. Jahangir Sarwar Khan, Junaid Alam, Hamid Hassan, Mohammad Iqbal;Pattern of intestinal obstruction a hospital based study; Pakistan Armed forces Medical J; issue year : 2007, issue number : 4, issue month : December
14. Natural history of patients with Adhesive small bowel obstruction; Br J Surgery 2000; 87; 1240-47

15. Small Intestinal Obstruction ; Surgical clinics of North America; 1997;67;597-620
16. M Beuran, I Ivanov, and MD Venter; Gallstone Ileus–Clinical and therapeutic aspects; J Med Life. 2010 November 15; 3(4): 365–370
17. Diagnosis and Management of Intestinal intussusception American Journal of Surgery; 1997;173;88-94
18. Lee SH, Ong ET. Changing pattern of intestinal obstruction in Malaysia; a review of 100 consecutive cases. Br J Surg 1991;78:181-2
19. Sarr MG, Bulkley GB, Zuidema GD. Preoperative recognition of intestinal strangulation obstruction: Prospective evaluation of diagnostic capability; *Am J Surg* 1983;145:176–181
20. Tim Jancelewicz & Lan T. Vu & Alexandra E. Shawo & Benjamin Yeh &Warren J. Gasper & Hobart W. Harris, Predicting Strangulated Small Bowel Obstruction: An Old Problem Revisited, J Gastrointest Surg (2009) 13:93–99

Appendix III - Master Chart

Sl. No	Name	Age	Sex	IP No	Diagnosis	Type of Obstruction	Acute/S/C	Bowel Condition	Previous Surgery	Pain	Tachycardia	Fever	Peritonitis	Leukocytosis	Altered Bowel Habits	Vomiting	Distension	Dehydration	Cause	I/L/E	Anatomic Part	Surgery done	Post Operative Complication
1	Subramani	65	M	46257	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Luminal	IL	Rectum	Resection Anastomosis	None
2	Semigammal	80	F	51152	Paraumbilical hernia with IO	NS	A	V	None	+	+	-	-	-	+	+	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
3	Raman	30	M	49978	Adhesive Intestinal Obstruction	NS	SA	V	None	-	-	-	-	-	+	+	-	-	TB Abdomen	EM	Ileum	Adhesiolysis	None
4	Asmath Begum	47	F	53610	Adhesive Intestinal Obstruction	NS	SA	V	Hysterectomy	+	+	-	-	-	+	-	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
5	Kali	70	M	53703	Ileal Thickening	NS	SA	V	None	+	-	-	-	-	+	-	+	-	TB Abdomen	IM	Ileum	Resection Anastomosis	None
6	Murugan	16	M	53862	Sigmoid Volvulus	S	A	NV	None	+	+	+	+	+	+	+	+	+	Volvulus	EM	Sigmoid Colon	Resection Anastomosis	Death
7	Raman Kumar	26	M	53884	Strangulated Inguinal Hernia	S	A	V	None	+	+	-	+	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
8	Rangaraj	59	M	53958	Perforated Adeno Ca Ileum	NS	A	V	None	+	+	-	+	+	-	+	+	+	Luminal	IL	Ileum	Resection Anastomosis	None
9	rajappan	50	M	54485	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	+	+	+	-	+	-	Extraluminal	EM	Ileum	Resection Anastomosis	Death
10	Jothimalar	25	F	54573	Adhesive Intestinal Obstruction	S	A	NV	Appendicectomy	+	+	+	+	-	+	+	+	+	Post Surgery	EM	Ileum	Resection Anastomosis	Wound Infection
11	Palani	50	M	57548	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	+	-	+	-	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
12	Manikandan	25	M	58384	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Luminal	IL	Rectum	Resection Anastomosis	None
13	Md. Nameem	18	M	58849	Ileal Stricture with perforation	NS	A	V	None	+	+	+	+	-	+	+	+	+	Extraluminal	IM	Ileum	Resection Anastomosis	None
14	Rayammal	52	F	59070	Adhesive Intestinal Obstruction	S	A	V	Hysterectomy	+	+	-	-	-	+	+	+	-	Post Surgery	EM	Ileum	Adhesiolysis	Burst Abdomen
15	Kuppammal	65	F	60562	Sigmoid Growth	NS	SA	V	None	-	-	-	+	-	+	-	+	-	Intraluminal	IL	Sigmoid Colon	Resection Anastomosis	None
16	Sekar	44	M	61144	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	+	+	-	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	None
17	Chinnammal	60	F	61484	Carcinoma Rectum	NS	SA	V	None	+	-	-	-	-	+	-	+	-	Intraluminal	IL	Rectum	Resection Anastomosis	None
18	Lannappan	61	M	62669	Gaint Inguinal Hernia	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Extraluminal	EM	Jejunum	Hernioplasty	None
19	mani	52	M	63202	Multiple Polyposis Coli	NS	SA	V	None	-	-	-	-	-	+	+	+	-	Luminal	IL	Descending Colon	Resection Anastomosis	None
20	Raju	18	M	64130	Obstructed Inguinal Hernia	NS	A	V	None	+	-	+	-	-	-	-	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
21	Jarchperdan	31	M	65477	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	+	+	-	+	Extraluminal	EM	Jejunum	Hernioplasty	None
22	Ramathal	70	F	65969	Adhesive Intestinal Obstruction	NS	A	NV	Hysterectomy	+	+	+	-	-	-	+	+	+	Post Surgery	EM	Ileum	Resection Anastomosis	None
23	Vellammal	39	F	65991	Strangulated Inguinal Hernia	S	A	V	None	+	+	-	-	-	+	+	+	-	Extraluminal	EM	Ileum	Hernioplasty	Wound Infection
24	Pandian	32	M	68173	Sigmoid Volvulus	S	A	NV	None	+	+	+	+	+	-	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Wound Infection
25	Venkatesh	39	M	70986	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	+	-	+	+	+	+	Extraluminal	EM	Ascending colon	Hernioplasty	Wound Infection
26	Geetha	33	F	74091	Ileocolic Intussusception with Adeno Ca	S	A	NV	None	+	+	+	+	+	-	+	+	+	Extraluminal	IL	Ileum	Resection Anastomosis	None
27	Chandran	55	M	79018	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	-	-	Extraluminal	EM	Ascending colon	Hernioplasty	None
28	Rakkammal	70	F	745	Adhesive Intestinal Obstruction	NS	SA	V	Hysterectomy	-	+	+	-	-	+	+	+	-	Post Surgery	EM	Ileum	Appendicectomy	None
29	Noorjehan	55	F	73907	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Luminal	IL	Rectum	Resection Anastomosis	None
30	Srirangammal	52	F	4375	Adhesive Intestinal Obstruction	NS	A	V	Hysterectomy	+	-	-	-	-	+	-	+	-	Post Surgery	EM	Jejunum	Adhesiolysis	Prolonged ileus
31	Saraswathi	48	F	6136	Incisional Hernia Obstructed	NS	A	V	Sterilization	+	+	-	+	-	+	+	+	+	Extraluminal	EM	Jejunum	Hernioplasty	Death
32	Chellammal	75	F	6498	Adhesive Intestinal Obstruction	NS	SA	V	Sterilization	-	+	+	-	-	+	-	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
33	Swamiappan	58	M	6692	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
34	Devendran	75	M	7000	Obstructed Inguinal Hernia	NS	A	V	Hernioplasty	+	-	-	-	-	+	-	+	+	Post Surgery	EM	Ileum	Hernioplasty	Prolonged ileus
35	Ganesan	48	M	8332	Adhesive Intestinal Obstruction	S	A	V	None	+	+	-	-	-	+	+	+	-	Appendicitis	EM	Ileum	Adhesiolysis	Wound Infection
36	Rajathi	36	F	8582	Adhesive Intestinal Obstruction	NS	A	V	Sterilization	+	-	-	-	-	-	+	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
37	Arumugam	80	M	9318	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	+	+	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
38	Usha Rani	35	F	8875	Adhesive Intestinal Obstruction	NS	SA	V	Sterilization	-	-	-	-	-	+	-	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
39	Muthusamy	65	M	10343	Carcinoma Rectum	NS	A	V	None	-	-	-	-	-	+	+	+	+	Intraluminal	IL	Rectum	Colostomy	None
40	Koorman	41	M	5461	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	+	+	+	+	+	+	Post Surgery	EM	Ileum	Resection Anastomosis	Prolonged ileus

Appendix III - Master Chart

Sl. No	Name	Age	Sex	IP No	Diagnosis	Type of Obstruction	Acute/S/C	Bowel Condition	Previous Surgery	Pain	Tachycardia	Fever	Peritonitis	Leukocytosis	Altered Bowel Habits	Vomiting	Distension	Dehydration	Cause	I/L/E	Anatomic Part	Surgery done	Post Operative Complication
41	Sundaram	48	M	10124	Adhesive Intestinal Obstruction	NS	SA	V	Hernioplasty	-	-	-	-	-	+	+	-	-	Post Surgery	EM	Jejunum	Laparoscopic Adhesiolysis	None
42	Kittan	44	M	16302	Strangulated Inguinal Hernia	S	A	NV	None	-	-	+	+	-	-	+	+	+	Peritonitis	EM	Ileum	Resection Anastomosis	Prolonged ileus
43	Senniyappan	32	M	11736	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	-	-	Extraluminal	EM	Ileum	Hernioplasty	Wound Infection
44	Sharma	41	M	5338	Adhesive Intestinal Obstruction	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Appendicitis	EM	Ileum	Appendicectomy	None
45	Ramaraj	50	M	11953	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	-	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
46	Nagarathinam	45	F	12004	Adhesive Intestinal Obstruction	NS	A	V	Appendicectomy	-	-	-	-	-	+	-	-	+	Post Surgery	EM	Ileum	Adhesiolysis	Prolonged ileus
47	Kumaresan	18	M	12021	Adhesive Intestinal Obstruction	NS	A	V	None	-	-	+	+	-	-	+	+	-	TB Abdomen	EM	Ileum	Adhesiolysis	None
48	Bhoominathan	72	M	11026	Adhesive Intestinal Obstruction	NS	SA	V	DU Perforation	-	-	-	-	-	+	+	+	-	Post Surgery	EM	Jejunum	Adhesiolysis	None
49	Lakshmi	80	F	12415	Adhesive Intestinal Obstruction	S	A	NV	Hysterectomy	+	+	+	+	+	+	+	+	+	Post Surgery	EM	Ileum	Resection Anastomosis	Death
50	Mahesh	22	M	12554	Adhesive Intestinal Obstruction	NS	SA	V	Appendicectomy	-	+	-	-	-	+	+	-	+	Post Surgery	EM	Ileum	Laparoscopic Adhesiolysis	None
51	Selvaraj	56	M	11159	Incisional Hernia	NS	SA	V	Perforation Closure	-	-	-	-	-	-	-	+	-	Extraluminal	EM	Jejunum	Hernioplasty	Wound Infection
52	Selvam	50	M	12890	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	+	-	+	+	-	-	Extraluminal	EM	Jejunum	Hernioplasty	None
53	Radha Krishnan	17	M	13182	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	+	-	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
54	Selvaraj	58	M	13616	Obstructed Inguinal Hernia	NS	A	V	Perforation Closure	+	-	-	-	-	-	+	+	-	Post Surgery	EM	Ileum	Hernioplasty	None
55	Saban Kumar	17	M	13458	Irreducible Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	-	+	-	Extraluminal	EM	Ascending colon	Hernioplasty	Wound Infection
56	nagaraj	41	M	13901	Internal Herniation	NS	A	NV	None	+	+	-	+	-	+	+	+	+	Extraluminal	EM	Jejunum	Resection Anastomosis	None
57	Pakeshwari	25	F	13482	Adhesive Intestinal Obstruction	NS	SA	V	None	+	-	+	-	-	+	+	-	-	TB Abdomen	EM	Ileum	Adhesiolysis	None
58	Maragathamani	45	F	15320	Adhesive Intestinal Obstruction	NS	A	V	CS	-	+	+	-	-	+	+	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
59	Natchammal	65	F	15755	Femoral Obstructed Hernia	S	A	V	None	+	+	+	+	-	+	+	+	+	Extraluminal	EM	Caecum Ileum	Hernioplasty	None
60	Lakshmanan	80	M	16483	Adhesive Intestinal Obstruction	NS	A	V	Perforation Closure	+	+	-	-	-	+	-	+	+	Post Surgery	EM	Jejunum	Adhesiolysis	None
61	Balan	51	M	16311	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	+	+	+	+	+	+	Extraluminal	EM	Sigmoid Colon	Resection Anastomosis	Wound Infection
62	Laxmanan	80	M	15814	Rectosigmoid Growth	NS	A	V	None	-	-	-	-	-	+	-	+	+	Luminal	IL	Sigmoid Colon	Colostomy	None
63	Seetha	21	F	16749	Adhesive Intestinal Obstruction	NS	A	V	Appendicectomy	-	-	-	-	-	-	-	+	-	Post Surgery	EM	Ileum	Adhesiolysis	Wound Infection
64	Chellammal	60	F	15939	Adhesive Intestinal Obstruction	NS	SA	V	Hysterectomy	+	-	-	+	-	+	+	+	+	Post Surgery	EM	Ileum	Adhesiolysis	Wound Infection
65	Ravi	46	M	16176	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Intraluminal	IL	Rectum	Resection Anastomosis	None
66	Pattan	63	M	18116	Epigastric Strangulated Hernia	S	A	V	None	+	+	+	+	-	-	+	+	-	Extraluminal	EM	Jejunum	Hernioplasty	None
67	Rajathi	38	F	16828	Adhesive Intestinal Obstruction	NS	SA	V	Sterilization	-	-	-	-	-	+	-	+	-	TB Abdomen	EM	Ileum	Adhesiolysis	None
68	Gurusamy	73	M	19572	Adhesive Intestinal Obstruction	NS	SA	V	None	+	+	-	-	-	-	+	+	-	TB Abdomen	EM	Ileum	Adhesiolysis	None
69	Thangaraj	60	M	19704	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	+	+	-	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Wound Infection
70	Krishnamoorthy	60	M	20538	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	-	+	Extraluminal	EM	Jejunum	Hernioplasty	None
71	Parvathammal	58	F	23916	Sigmoid Growth	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Luminal	IL	Sigmoid Colon	Resection Anastomosis	None
72	Srinivasan	29	M	24493	Strangulated Inguinal Hernia	S	A	V	None	+	+	-	+	-	-	+	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
73	Shek Hussain	73	M	26055	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	+	+	-	+	Extraluminal	EM	Descending Colon	Hernioplasty	None
74	Nagamani	41	F	26317	Adhesive Intestinal Obstruction	NS	SA	V	CS	+	-	+	-	-	-	+	-	+	Post Surgery	EM	Ileum	Laparoscopic Adhesiolysis	None
75	karuppusamy	63	M	26422	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	+	+	+	-	Extraluminal	EM	Jejunum	Hernioplasty	None
76	Nagaraj	65	M	26541	Umbilical Strangulated Hernia	S	A	NV	None	+	+	-	+	+	-	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Wound Infection
77	Kasi	70	F	27701	Adhesive Intestinal Obstruction	NS	A	V	Sterilization	-	+	+	-	-	+	+	-	-	Post Surgery	EM	Omentum Ileum	Adhesiolysis	None
78	Ravi	33	M	27619	Adhesive Intestinal Obstruction	NS	SA	V	None	-	-	-	-	-	-	-	+	+	Appendicitis	EM	Ileum	Appendicectomy	None
79	Subramani	50	M	26906	Incisional Hernia	NS	SA	V	Blunt Injury Abdomen	+	-	-	-	-	-	-	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
80	Muruganandam	34	M	28397	Strangulated Inguinal Hernia	S	A	V	None	+	+	+	+	-	+	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None

Appendix III - Master Chart

Sl. No	Name	Age	Sex	IP No	Diagnosis	Type of Obstruction	Acute/S/C	Bowel Condition	Previous Surgery	Pain	Tachycardia	Fever	Peritonitis	Leukocytosis	Altered Bowel Habits	Vomiting	Distension	Dehydration	Cause	I/L/E	Anatomic Part	Surgery done	Post Operative Complication
81	Venugopal	54	M	28892	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	-	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
82	Alla Basha	55	M	30013	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	-	-	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
83	Raja Sulochana	48	F	30191	Adhesive Intestinal Obstruction	NS	A	V	cs	+	+	-	-	-	-	-	+	+	Post Surgery	EM	Ileum	Adhesiolysis	None
84	Mohan	56	M	30396	Ileal Stricture	NS	SA	V	None	+	+	-	-	+	-	+	+	-	Mural	IM	Ileum	Resection Anastomosis	None
85	Kosalaimuthu	34	M	31064	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	+	+	-	Extraluminal	EM	Ascending colon	Hernioplasty	None
86	Arichami	55	M	30698	Adhesive Intestinal Obstruction	NS	SA	V	None	-	+	+	-	-	-	+	+	-	Pancreatitis	EM	Small Omentum	Adhesiolysis	None
87	Padmavathy	50	F	31772	Obstructed Inguinal Hernia	NS	A	V	None	+	-	+	-	-	+	-	+	+	Extraluminal	EM	Ileum	Hernioplasty	Wound Infection
88	Selvaraj	20	M	32481	Adhesive Intestinal Obstruction	NS	SA	V	Perforation Closure	-	-	-	-	-	+	-	+	-	Post Surgery	EM	Ascending colon	Adhesiolysis	Faecal Fistula
89	Kittusamy	51	M	34334	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	-	+	+	Extraluminal	EM	Caecum	Hernioplasty	Wound Infection
90	Muruganathan	22	M	34566	Adhesive Intestinal Obstruction	NS	A	V	Appendicectomy	+	+	-	-	-	-	+	+	+	Post Surgery	EM	Ileum	Adhesiolysis	None
91	Sangottian	50	M	34895	Adhesive Intestinal Obstruction	NS	SA	V	Hernioplasty	-	-	-	-	-	+	-	-	-	Post Surgery	EM	Ileum	Adhesiolysis	Wound Infection
92	Aaraiammal	70	F	35681	Adhesive Intestinal Obstruction	S	A	NV	Hysterectomy	+	+	+	+	+	-	+	+	+	Post Surgery	EM	Ileum	Resection Anastomosis	Wound Infection
93	Parameshwari	50	F	35684	Adhesive Intestinal Obstruction	S	A	NV	CS	+	+	-	+	+	+	+	+	+	Post Surgery	EM	Ileum	Resection Anastomosis	None
94	Ravichandran	50	M	36761	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
95	Valliammal	43	F	37053	Adhesive Intestinal Obstruction	NS	SA	V	Sterilization	-	-	-	-	-	-	+	-	-	Post Surgery	IM	Ileum	Adhesiolysis	None
96	Maheshwari	52	F	38143	Incisional Strangulated Hernia	S	A	NV	None	+	+	+	+	+	+	-	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Death
97	Chinnarajan	65	M	38988	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
98	Arukkani	57	F	38569	Umbilical Strangulated Hernia	S	A	V	None	+	+	-	+	+	+	+	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
99	Sundaram	62	M	39090	Adhesive Intestinal Obstruction	NS	SA	V	DU Perforation	-	-	-	-	-	+	-	-	+	Post Surgery	EM	Jejunum	Adhesiolysis	None
100	Mani	42	M	39569	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	+	-	Extraluminal	EM	Ascending colon	Hernioplasty	None
101	Subramani	60	M	39665	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	+	+	+	-	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
102	Arumugam	46	M	40193	Umbilical Strangulated Hernia	S	A	NV	None	+	+	+	-	+	+	+	+	+	Extraluminal	EM	Jejunum	Resection Anastomosis	None
103	Maheshwari	55	F	41578	Adhesive Intestinal Obstruction	NS	A	V	Hysterectomy	-	+	-	+	-	-	+	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
104	Kaliammal	57	F	41658	Incisional Strangulated Hernia	S	A	V	Hysterectomy	+	+	-	+	-	+	+	+	+	Extraluminal	EM	Jejunum	Hernioplasty	Wound Infection
105	Saravanam	30	M	43031	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	-	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
106	Kanagasamy	35	M	43194	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	-	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
107	Saroja	40	F	43524	Femoral Obstructed Hernia	NS	A	V	None	+	-	-	+	-	+	+	-	+	Extraluminal	EM	Ileum	Hernioplasty	Wound Infection
108	Krishnan	65	M	43571	Adhesive Intestinal Obstruction	S	A	NV	None	+	+	-	+	+	-	+	-	+	Pancreatitis	EM	Ileum	Resection Anastomosis	Wound Infection
109	thangavel	52	M	43634	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	-	+	+	+	Extraluminal	EM	Jejunum	Hernioplasty	None
110	Ayyasamy	50	M	44489	Ileal Stricture	NS	SA	V	None	+	+	-	-	-	+	-	+	-	Extraluminal	IM	Ileum	Resection Anastomosis	None
111	Karthik	18	M	45054	Adhesive Intestinal Obstruction	NS	A	V	None	+	+	-	-	-	-	-	+	+	Appendicular Abscess	EM	Ileum	Appendicectomy	Prolonged ileus
112	Shanmugam	35	M	45108	Incisional Strangulated Hernia	S	A	V	Perforation Closure	+	+	+	+	+	+	+	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
113	Maheshwari	40	F	45460	Adhesive Intestinal Obstruction	S	A	V	CS	+	-	+	+	-	-	+	+	+	Post Surgery	EM	Ileum	Adhesiolysis	None
114	Venkatesh	65	M	44169	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Luminal	IL	Rectum	Resection Anastomosis	None
115	Sasikumar	29	M	45695	Adhesive Intestinal Obstruction	NS	A	V	Appendicectomy	+	-	+	-	-	-	-	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
116	Rangasamy	65	M	45513	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	+	-	+	-	-	+	Extraluminal	EM	Sigmoid Colon	Hernioplasty	None
117	Kumar	23	M	45943	Adhesive Intestinal Obstruction	NS	A	V	None	+	-	-	-	-	-	+	+	+	Appendicitis	EM	Ileum	Appendicectomy	None
118	Arumugam	65	M	46565	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	-	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
119	Arunachalam	42	M	47320	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	-	+	-	Extraluminal	EM	Jejunum	Hernioplasty	None
120	Padma	40	F	47457	Incisional Strangulated Hernia	S	A	V	Sterilization	+	+	-	+	+	+	+	+	+	Extraluminal	EM	Ascending colon	Hernioplasty	Wound Infection

Appendix III - Master Chart

Sl. No	Name	Age	Sex	IP No	Diagnosis	Type of Obstruction	Acute/S/C	Bowel Condition	Previous Surgery	Pain	Tachycardia	Fever	Peritonitis	Leukocytosis	Altered Bowel Habits	Vomiting	Distension	Dehydration	Cause	I/L/E	Anatomic Part	Surgery done	Post Operative Complication
121	mani	50	M	46840	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	-	+	+	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Wound Infection
122	Kumarasamy	42	M	48211	Adhesive Intestinal Obstruction	NS	SA	V	Appendicectomy	-	-	-	-	-	-	+	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
123	Dhanalaxmi	70	F	48326	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	+	-	-	Intraluminal	IL	Rectum	Colostomy	None
124	Gopal	65	M	48962	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	-	-	Extraluminal	EM	Jejunum	Hernioplasty	None
125	Subburam	58	M	48984	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
126	Velammal	65	F	46209	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Intraluminal	IL	Rectum	Resection Anastomosis	None
127	Ponnusamy	62	M	49235	Adhesive Intestinal Obstruction	NS	SA	V	DU Perforation	+	-	+	-	-	-	+	-	+	Post Surgery	EM	Jejunum	Adhesiolysis	Wound Infection
128	Mallammal	70	F	46272	Adhesive Intestinal Obstruction	NS	SA	V	Hysterectomy	+	+	-	-	-	+	-	+	-	Post Surgery	EM	Ileum	Adhesiolysis	None
129	Ponnusamy	65	M	51238	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	+	-	-	+	Extraluminal	EM	Jejunum	Hernioplasty	Wound Infection
130	Thirumoorthy	50	M	51246	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	+	-	-	-	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
131	Thirumoorthy	50	M	49959	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	+	+	+	+	+	+	Extraluminal	EM	Jejunum	Resection Anastomosis	Prolonged ileus
132	Karthik	23	M	53540	Adhesive Intestinal Obstruction	S	A	NV	None	+	+	+	+	+	+	+	+	-	Peritonitis	EM	Jejunum	Resection Anastomosis	Wound Infection
133	Chellammal	80	F	51398	Carcinoma Caecum	NS	SA	V	None	-	-	-	-	-	+	-	+	-	Luminal	IL	Caecum	Resection Anastomosis	None
134	Parvathammal	65	F	47619	Ca Sigmoid Colon	NS	A	V	None	+	+	-	-	-	-	+	+	+	Luminal	IL	Sigmoid Colon	Colostomy	None
135	Poovammal	68	F	61033	Incisional Irreducible Hernia	NS	A	V	Sterilization	+	-	+	-	-	-	+	+	-	Extraluminal	EM	Ileum	Hernioplasty	None
136	Badhal	20	M	57590	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	-	-	Extraluminal	EM	Descending Colon	Hernioplasty	Wound Infection
137	Venkatan	76	M	58396	Strangulated Inguinal Hernia	S	A	NV	None	+	+	-	+	+	+	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Death
138	Arumugam	55	M	58582	Irreducible Inguinal Hernia	NS	SA	V	None	+	-	-	-	-	-	+	-	+	Spontaneous	EM	Jejunum	Hernioplasty	None
139	ramachandran	45	M	63954	Irreducible Inguinal Hernia	NS	SA	V	None	+	-	-	-	-	+	+	-	-	Extraluminal	EM	Ileum	Hernioplasty	None
140	Amal Raj	41	M	63695	Obstructed Inguinal Hernia	NS	A	V	None	+	+	-	-	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
141	Mani	68	M	63865	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	+	-	+	+	+	-	Extraluminal	EM	Jejunum	Hernioplasty	None
142	Mayasam	66	M	64074	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	-	-	+	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
143	karuppusamy	35	M	64127	Obstructed Inguinal Hernia	NS	A	V	None	+	-	-	+	-	+	-	-	+	Extraluminal	EM	Ileum	Hernioplasty	None
144	Somashekar	30	M	62345	Adhesive Intestinal Obstruction	NS	SA	V	None	+	-	-	-	-	+	-	+	-	TB Abdomen	IM	Ileum	Adhesiolysis	Wound Infection
145	Narayanan	50	M	64874	Obstructed Inguinal Hernia	NS	A	V	None	-	-	-	-	-	+	-	+	-	Post Surgery	EM	Ileum	Hernioplasty	Faecal Fistula
146	Muthuveeran	45	M	65011	Adhesive Intestinal Obstruction	NS	A	V	None	+	+	-	-	-	-	-	+	+	Appendicitis	EM	Ileum	Appendicectomy	Wound Infection
147	Kannan	31	M	66929	Strangulated Inguinal Hernia	S	A	NV	None	+	+	-	+	+	+	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Prolonged ileus
148	Ravi	35	M	67205	Adhesive Intestinal Obstruction	NS	A	V	None	+	-	-	-	-	-	+	+	-	Spontaneous	EM	Jejunum	Adhesiolysis	None
149	Lakshmi	69	F	67001	Adhesive Intestinal Obstruction	NS	SA	V	Incisional Hernia	-	-	-	-	-	+	-	+	-	Post Surgery	EM	Jejunum	Adhesiolysis	None
150	Sundaram	61	M	67324	Carcinoma Rectum	NS	SA	V	None	-	+	-	-	-	+	-	+	-	Intraluminal	IL	Rectum	Resection Anastomosis	None
151	Radhamalli	16	F	65905	Adhesive Intestinal Obstruction	NS	SA	V	Ileal Perforation	-	-	-	-	-	-	+	+	+	Post Surgery	EM	Descending Colon	Laparoscopic Adhesiolysis	None
152	Nataraj	62	M	61608	Carcinoma Rectum	NS	SA	V	None	-	-	-	-	-	+	+	-	-	Intraluminal	IL	Rectum	Resection Anastomosis	None
153	Gopala Swamy	57	M	68814	Obstructed Inguinal Hernia	NS	A	V	None	-	-	-	-	-	-	+	+	+	Extraluminal	EM	Ileum	Hernioplasty	None
154	Narayanasamy	48	M	69264	Strangulated Inguinal Hernia	S	A	NV	None	+	+	+	-	+	+	+	+	+	Extraluminal	EM	Ileum	Resection Anastomosis	Wound Infection

Appendix IV

Key to Master Chart

1. M – Male
2. F – Female
3. S – Strangulating
4. NS – Nonstrangulating
5. A – Acute
6. SA – Subacute
7. V – Viable
8. NV – Nonviable
9. TB – Tuberculosis
10. IL – Intraluminal
11. IM – Intramural
12. EM – Extramural
13. Ca - Carcinoma